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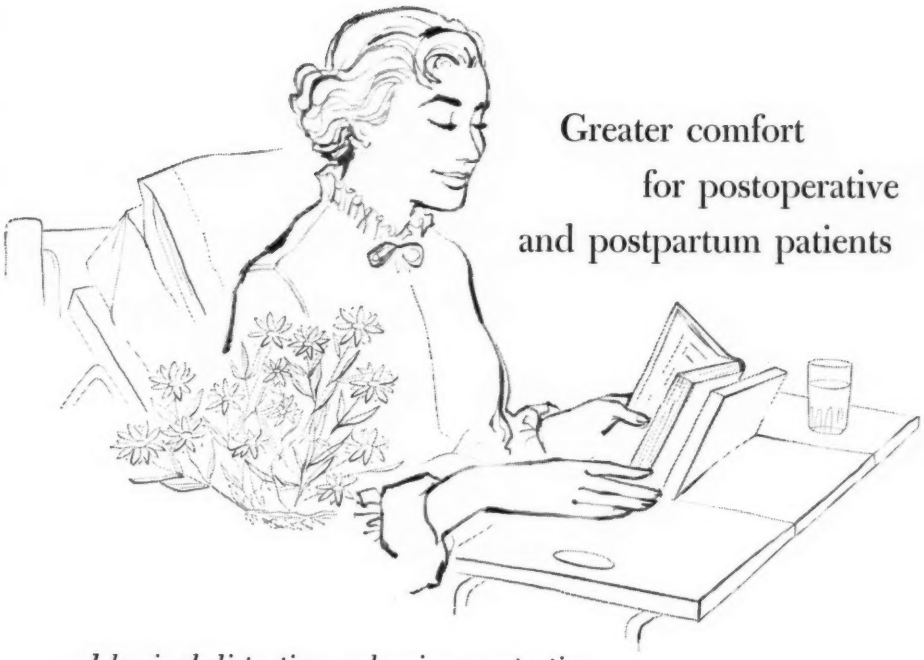

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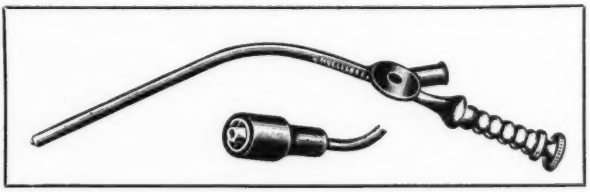

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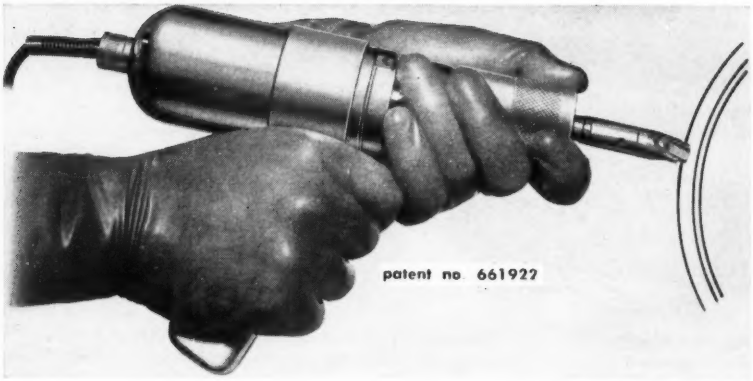

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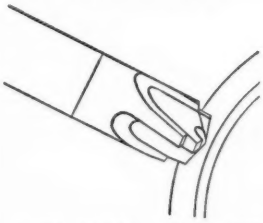

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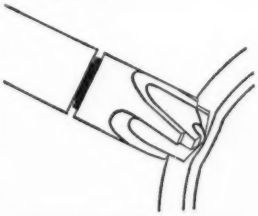

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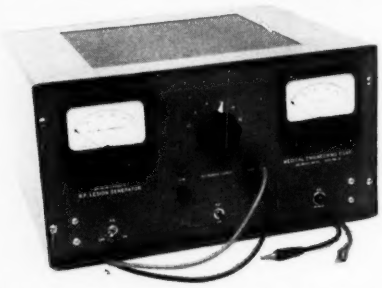

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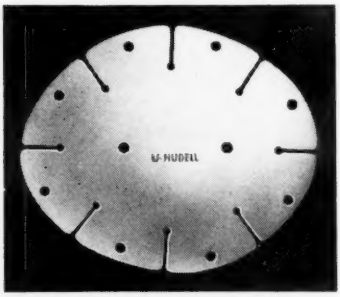

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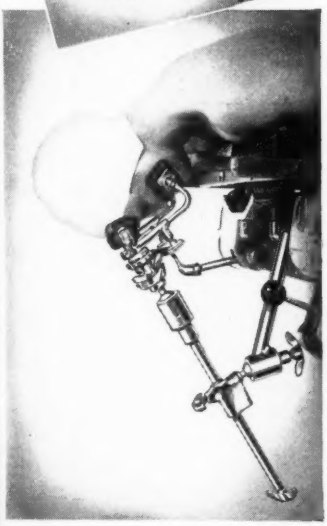

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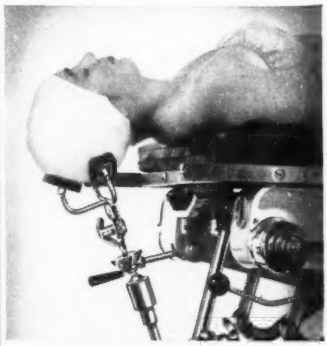

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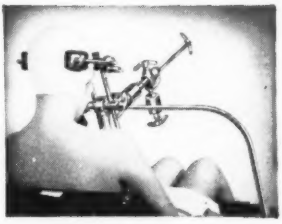

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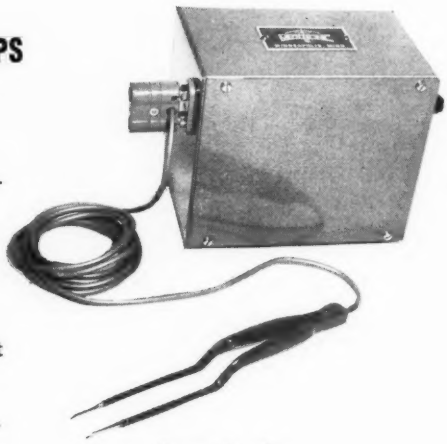

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THE odontoid process of the axis owes its importance to its anatomical location high in the cervical spine. Since fractures of this small process are associated with dislocations of the atlas on the axis, which in turn compress the upper cervical spinal cord, it is not surprising that both morbidity and mortality are high with these injuries. Geoffrey Jefferson[21] in 1920 and Osgood and Lund[28] in 1928 surveyed 55 of the fractures of the odontoid process reported to that time, the mortality being about 50 per cent. In at least 10 of the fatal cases reviewed by Osgood and Lund death was not immediate but occurred in patients who had become increasingly disabled over weeks, months, or years. Possibly a tendency to report fatal cases at that time made the mortality more apparent than real. By 1945 Hinchey and Bickel[20] surveyed the previous literature and added 8 cases of their own, making a total of 120.

Osgood and Lund[28] in 1928 discussed a fracture of the odontoid process received by Dr. Lund himself in 1925. His injury was the forerunner to the great increase in the number of fractured odontoid processes reported since then. Dr. Lund's accident occurred in a Model-T Ford; the tremendous advances in power and speed of automobiles since 1928 are a matter of record. As a result there is an increase in fractures and dislocations of the upper cervical spine associated with severe trauma. Undoubtedly the increasing use of roentgenograms taken of areas in which injury is suspected results in more frequent recognition of abnormalities in the cervical region.

Embryologically the odontoid process arises as a part of the body of the atlas but later fuses with the upper portion of the body of the axis and articulates with the posterior surface of the small body of the atlas. It is held firmly in place dorsally by the transverse ligament of the atlas. In about 23 per cent of people 30 to 50 years old[30] there is an incomplete union of the odontoid process with the axis. A small island of cartilage, the vestige of the original intervertebral disc, remains at the base of the odontoid process. This island of cartilage in some cases may contribute to the ease with which dislocations of the odontoid process occur. Under certain circumstances, as in chronic rheumatoid arthritis and in acute pyogenic infec-

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tions around the neck, as well as in trauma, the transverse ligament may rupture, allowing forward dislocation of the atlas on the axis.[24] Congenital absence of the odontoid process with excessive mobility of the atlas on the axis has been reported a few times.[17,26,35,37]

Reliable information is not available concerning the frequency of trauma to the upper cervical spine as the cause of immediate death. Life is incompatible with a severe crushing injury to the cervical spinal cord at the level of the atlas and axis; hence, if fractures of the odontoid process cause damage to the spinal cord, such injured patients do not ordinarily come under medical surveillance. The majority of surviving patients with acute fracture of the odontoid process have little or no neurologic disability, and, except for the frequency of associated injuries, the mortality from these lesions, if properly treated, should be negligible. Many patients actually walk into the emergency room.

Plaut,[31] Schwarz and Wigton,[36] and others maintain that solid healing of a fractured odontoid process does not occur. Osgood and Lund[28] stated that 2 patients with such fractures, who died of intercurrent disease, showed firm union of the odontoid process at autopsy. Cone and Turner[9] reported that 1 patient on whom they performed a fusion of the upper cervical spine displayed firm union of the fractured odontoid process several months later.

Hinchey and Bickel,[20] however, believed that fractures of the odontoid process rarely unite but there is usually no resultant disability. Cone and Turner[9] and Osgood and Lund[28] thought that difficulties commonly arise when there is failure of proper union of these fractures. In 1948 Crutchfield[13] wrote that fractures of the odontoid process with dislocation do not heal satisfactorily and advised cervical fusion; later[12] he treated his patients conservatively by maintaining skeletal traction for several weeks, then by using rigid support for about 6 or 8 months.

More recently Amyes and Anderson[2] surveyed 63 cases of fractures of the odontoid process seen at Los Angeles County Hospital and in their private practice. Their patients were treated by various methods, some by use of skeletal traction and others by application of plaster cast to the neck. They used no set form of therapy in the group, although a rigid steel-reinforced leather collar was recommended after traction was discontinued—"... there was a great deal of variation in the length of time the neck support was worn. Each patient was instructed to continue wearing his brace until healing was demonstrated by x-ray." Although they advised follow-up for at least a year, they apparently lost sight of many of their patients before a year had passed. They did not record any late complications, and they considered that only 3 of their patients needed cervical fusion for stabilization of the fracture-dislocation. They frequently used laminagraphs, both to confirm the fracture of the odontoid process and to determine the progress of healing.

Undoubtedly some fractures of the odontoid process do heal solidly when patients are treated by maintaining skeletal traction for 3 months,

followed by their wearing a rigid external support for another 3 to 6 months. These cases have been reported and there is at least 1 in our series (Fig. 1).

The literature is replete with reports of late complications of nonunited fractures of the odontoid process, chiefly recurrent stiffness of the neck, episodes of complete or partial tetraplegia resulting from relatively minor trauma, and eventually production of a picture of chronically progressive compression of the high cervical spinal cord.[3,38] Frequently death has resulted from mild secondary trauma as in the case reported by Austin Flint, Sr., in 1869.[16] The most dramatic report is that of Elliot and Sachs[14] of a man who survived four episodes of compression of the spinal cord with varying degrees of paralysis over a period of almost 35 years until he died from compression of the upper spinal cord. His cervical spine and spinal cord were studied by Dr. Ernest Sachs and the cause of death—the fracture-dislocation of the odontoid process with compression of the spinal cord—was illustrated in their publication in 1912. Mixter and Osgood,[25] Pilcher,[29] Wilson,[40] Rogers,[34] Cone and Turner,[9,39] Kamman,[23] Kahn and Yglesias,[22] Craig,[10] Schwarz and Wigton,[36] Brookes,[7] Jefferson,[21] Blockey and Purser,[5] and Alexander *et al.*[1] presented similar stories of patients who showed serious neurologic disorders or who died as the result of nonunited fractures of the odontoid process.

Cone and Turner,[9] and Gallie,[18] concluded many years ago that all patients with fractures of the odontoid process should be treated by performing open operation and cervical fusion. Their results were excellent and the mortality was low. A similar policy was followed with some of the patients presented in this paper; others, for various reasons, were treated conservatively. A modification of operative procedure and other significant details in the management of these cases are considered sufficiently important to warrant further emphasis.

Although there has been no special reference to it in the medical literature, our experience gained in a few cases led us to the tentative conclusion that fractures of the odontoid process that break well into the body of the axis rather than breaking at the base of the odontoid process may heal satisfactorily without operative fusion if reduction by traction is accomplished.

Several surgical approaches to fractures of the odontoid process have been made in the last 60 years. Church and Eisendrath[8] in 1892 and Brokaw[6] in 1898 described methods of fixation of fractures of the spine by tying spinous processes and laminae together with various materials, including silk, silkworm gut, and kangaroo gut. Their publications did not deal with fractures or dislocations high in the cervical spine. Later Mixter and Osgood[25] in 1910 and Ely[15] in 1911 reported securing dislocations of the atlas and axis together with heavy silk sutures under the lamina of C1 and around the spinous process of C2.

With improved roentgenographic techniques making easier recognition

of fracture-dislocations and the treatment more certain of success, most of these lesions—particularly those discovered soon after injury—were treated by manipulation, brief traction, and immobilization with plaster cast. The report of Osgood and Lund[28] is an excellent example of such successful treatment. The popularity of manipulative reductions and immobilization with plaster cast attended by few complications, is attested to by the publications of Brookes[7] and Roberts.[32,33] They suggested, however, that chronic dislocations of the upper cervical spine might be treated better with surgical fusion.

Nevertheless, as laminectomy became a well recognized procedure, carried out with low mortality, the immediate problem of compression of the spinal cord was attacked directly by performing laminectomy and suboccipital craniectomy. A case was described by Craig[10] in which the patient's chronic neurologic disability was much improved by decompression without fusion. Kahn and Yglesias,[22] and Rogers,[34] also decompressed the spinal cord by performing laminectomy and enlarging the foramen magnum, but at the same time they did an extensive fusion of the cervical spine to the occiput. One of the early cases of Turner and Cone[9,39] was handled in a similar way with success.

With the introduction of skeletal traction advocated by Crutchfield,[11,12] and by Barton,[4] by inserting metal tongs into the skull, the ease of reducing dislocations of the atlas on the axis became apparent—even when done months or years after injury.[7]

A satisfactory regime for treatment of these patients was outlined with various detailed modifications by Cone and Turner,[9] Gallie,[18,19] Rogers,[34] and Winant.[41] After the fracture-dislocation is confirmed by a satisfactory roentgenogram, Crutchfield tongs are applied and the dislocation is reduced. The general condition of the patient dictates the time to do a surgical fusion, following reduction of the dislocation. If the patient has no associated injuries and his general condition is satisfactory, early fusion is done in the first days or weeks after injury without decompressive laminectomy. After healing of the wound, the patient is allowed to be up with external support of plaster cast or a brace. It is doubtful if the late neurologic complications of chronic atlo-axial dislocation are so often caused by callus described in the older literature as by the excessive mobility of the upper cervical spine which repeatedly injures the cervical spinal cord. There is, therefore, no logical rationale for not treating the chronic lesions by reduction and fusion as done in the acute cases.

The methods of fixation of atlo-axial dislocations have varied widely. If a laminectomy of the atlas has been done previously, a more complex method of fusion is necessary: the lateral facets are utilized and the occiput is often fused to the lower cervical spine. Cone and Turner,[9] Winant,[41] Kahn and Yglesias,[22] and others, wired fragments of bone between the occiput and the atlas and/or axis. Gallie used a simple fusion of the arch of the atlas to the arch of the axis.[18]

ANALYSIS OF CASE MATERIAL

Twenty-five patients are in the group, ranging in age from 21 months to 75 years. There were 22 men, 2 women and 1 child. Sixteen of the injuries were the direct result of motor-vehicle accidents; 8 patients were drivers, 6 were passengers, and it is uncertain whether or not 2 patients were drivers or passengers. Three of the injured were asleep at the time of the accident. Falls from a height were the cause of 5 of the fractures; 1 patient was thrown against a building by a cow; 1 fracture was the result of metastasis of a prostatic carcinoma to the body of the axis. The 21-month-old infant either had trauma to the neck at birth or an infection about the neck.[1] One fracture appeared spontaneously in association with severe rheumatoid arthritis.

DEATHS

(1) *#114489.* A 21-month-old child died the day after decompressive laminectomy was done under local anesthesia with the aid of electrophrenic stimulation. At necropsy the cervical spinal cord was observed to be only a ribbon.[1]

(2) *#167647.* A 58-year-old man with metastasis of a prostatic carcinoma to the atlas died on the 14th day after operative fusion of the occipitocervical region. However, he died of a pulmonary embolus and no neurologic deficit was noted either before or after surgery.

(3) *#168587.* A 70-year-old man with severe tetraparesis died at home 9 months after injury, apparently of complications of the urinary tract. He was treated by use of skeletal traction with Crutchfield tongs for 25 days in the hospital, then by rest in bed at home. His physician reported that he showed slow but progressive improvement in neurologic function during the 9 months he survived. No surgical procedure was performed.

(4) *#176053.* A 47-year-old man with severe cerebral contusion, acute subdural hematoma, a fracture of one hip, and an injury of the brachial plexus, died about 9 months after injury in another hospital. The fracture of the odontoid process had been treated by the use of skeletal traction in the hospital and by rest in bed elsewhere. The amount of recovery of neurologic function was uncertain because of his prolonged coma from injuries of the head. No surgery was performed for the fractured odontoid process.

(5) *#231761.* A 23-year-old man was in an automobile accident in which he received severe cerebral contusion, an injury of the chest requiring thoracostomy and tracheostomy, and other injuries. Roentgenograms showed fracture-dislocation of the odontoid process which was reduced by traction applied with Crutchfield tongs. The patient lived for 13 days in a state of decerebrate rigidity, never regaining consciousness. No operative treatment of the fractured odontoid process was carried out. At necropsy no compression of the cervical spinal cord was seen and his death was attributed to the head injuries.

Comment. There is one lesson to be learned from studying these 5 deaths: The infant treated by removal of the posterior rim of the foramen magnum at laminectomy would have been treated more satisfactorily by application

of skeletal traction in order to reduce the dislocation. Then, if neurologic improvement had occurred, possibly a fusion could have been performed later. The difficulty in recognizing the normal configuration of the cervical spine in the child[1] led the surgeon to suppose that he was dealing with a neoplasm rather than with an anterior dislocation of the atlas on the axis.

SURVIVING PATIENTS TREATED CONSERVATIVELY

There were 4 patients treated by traction followed by further rigid immobilization for varying lengths of time. In all of these, followed from 10 months to 6 years, the neurologic function remained normal.

FIG. 1. Multiple injuries including fracture-dislocation of odontoid process treated by prolonged skeletal traction. Roentgenograms taken 6 years later show solid healing with slight malalignment.

The late follow-up studies in 3 patients are sufficiently complete to be valuable in this investigation. The fourth patient has moved to another state and was returning to work when last heard from 10 months after his injury.

(1) *#139842.* A 75-year-old man with fracture of the odontoid process and dislocation of the atlas on the axis was treated by application of skeletal traction and rest in bed. The urinary bladder became obstructed associated with benign prostatic hypertrophy and azotemia. The obstruction was not relieved by drainage with catheter until he was allowed to get out of bed. He was able to walk with a modified brace[27] to which traction was applied through two sets of Crutchfield tongs for 28 days. The patient wore his brace only sporadically after removal of the tongs, and he has not wanted further treatment. There was excessive mobility of the atlas on the axis with nonunion of the fractured odontoid process. No neurologic deficit was present but he suffered from some stiffness and discomfort of the neck.

(2) *#100277.* A 31-year-old man was injured July 15, 1949. In addition to a fracture of the odontoid process, he had multiple fractures of the mandible, cerebral contusion with prolonged coma lasting several weeks, a fracture of the right radius and ulna, and contusion of the right lung and right kidney. Of necessity he was treated by using skeletal traction with Crutchfield tongs for 10 weeks on a Foster turning frame. He was immobilized in bed without traction for several weeks thereafter without other support to his neck. Roentgenograms of his cervical spine 6 years later showed excellent solid bony healing of the fracture of the odontoid process although it was displaced slightly forward to the right (Fig. 1). There were arthritic changes in the atlo-occipital joints and in the atlo-axial joints. Motion of his neck was moderately limited and somewhat painful but there was no neurologic deficit.

Comment. This is a striking example of a fractured odontoid process that has healed without fusion, but it is the only one in this series that has definitely done so. The patient also showed solid union in malposition of the fractures of the right radius and ulna and of the multiple fractures of the mandible. Satisfactory reduction of the fractures was necessarily delayed for several weeks because of his other injuries and the seriousness of his general condition.

(3) *#234418.* A 29-year-old Negro was injured when he fell down a flight of stairs on June 6, 1956. He suffered with pain in his neck, and although he is not clear about incidents immediately following the accident, he thought that his right side was very weak for several hours afterwards.

He reported to the emergency room of a hospital where roentgenograms showed a fracture of the odontoid process visible only in the anteroposterior view, with no displacement and no evidence of a fracture in the lateral view. Halter traction was applied with about 7 pounds' weight for 8 days, followed by a "cast," the extent of which is not known, for 18 days. Since he had residual stiffness of the neck following this treatment, he was referred to a neurosurgeon for the first time on Sept. 21, 1956.

At that time his neck showed some stiffness; the neurologic findings were considered to be within normal limits. Roentgenograms taken in the lateral projection of the upper cervical spine in flexion and extension revealed movement of about 6 mm. forward of the atlas on the axis in forward flexion. He was, therefore, referred to a veterans' hospital for fusion of the cervical spine at his request because of financial circumstances. There, roentgenograms were said not to show any instability of the fracture, and he was discharged without operation or external support to his neck.

Because of our interest in this problem and in following the patient, he was seen again on April 5, 1957. He had no physical complaints and was working in a restaurant. He admitted that there was some limitation of movements of his neck but denied weakness of his extremities or other significant symptoms.

Examination showed about 30 per cent limitation of flexion and extension of the neck, about 50 per cent limitation of rotation of the head, and about 60 per cent limitation of lateral bending of the head. The right biceps reflex was more active than the left, the left knee jerk was more active than the right, but there were no other abnormalities.

Roentgenograms of the cervical spine revealed continued evidence of the fracture of the odontoid process at its base in the anteroposterior view, but the most striking finding was marked forward dislocation of the atlas on the axis of about 1 cm. in forward flexion, with tilting forward of the atlas (Fig. 2). The patient was advised to have operative fusion of the cervical spine because of the instability, but to this date has not made a decision.

SURVIVING PATIENTS UNTREATED

There were 2 patients who had no specific treatment—immobilization, traction, or surgery—for fracture of the odontoid process:

(1) *#214500.* A 36-year-old man was in an automobile accident in which he thinks his head was thrown against the rear-view mirror above the windshield. He had severe stiffness and limitation of motion of his neck but returned to work

FIG. 2. (*Left*) Lateral film of cervical spine 10 months after injury. Note forward dislocation of atlas on axis. (*Right*) Laminagraph of odontoid process in lateral view without flexion or extension 11 months after injury. Original films showed no displacement in lateral view; fracture visible only on anteroposterior "open-mouth" view.

FIG. 3. Roentgenogram in lateral projection 2 years after fracture-dislocation of odontoid process without treatment. There is poor alignment and movement of C1 on C2.

several weeks after the accident. He was able to do only limited physical work, however, because of pain in his neck. He was first seen at the North Carolina Baptist Hospital 2 years after his injury. There was limitation of all the motions of his back and he complained of a "popping" sensation of his neck on certain movements. There was no weakness of the arms or legs and the neurologic findings were considered to be normal.

Roentgenograms of the cervical spine disclosed a nonunited fracture of the odontoid process. Lateral films made in flexion and extension of the neck revealed anterior dislocation of C1 on C2 with further movement forward on flexion of the neck (Fig. 3). The body of the

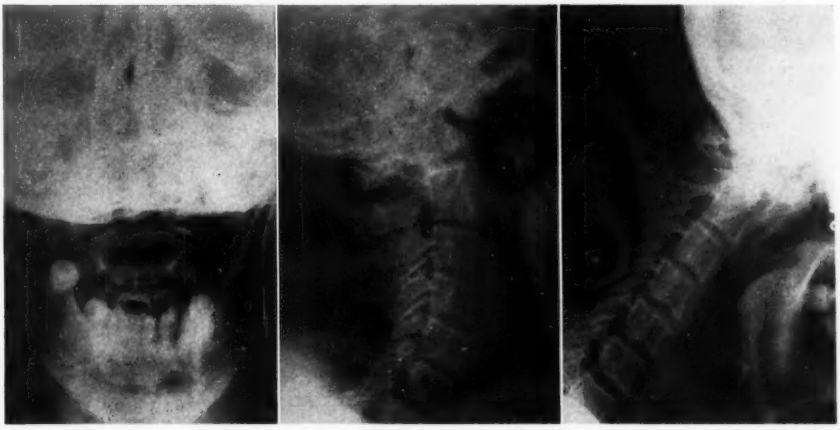


FIG. 4. Anteroposterior and lateral roentgenograms, the lateral films taken in flexion and extension 5½ years after fracture-dislocation of odontoid process treated by brace for 3 months after injury. Fracture still evident and there is excessive mobility of C1 on C2.

atlas protruded into the posterior pharyngeal shadow of air on forward flexion of the neck. He refused hospitalization for surgical fusion of the upper cervical spine.

(2) *#115136.* A 34-year-old man was examined first 3 months after an injury which occurred when the trailer-truck he was driving jack-knifed, pinning him in the cab of the truck. He complained of stiffness of his neck but no other serious disability. Three months later roentgenograms showed a fracture of the odontoid process. A brace was applied to his neck which he wore intermittently for several months. He returned to his occupation of driving a truck and, with the exception of fairly frequent headaches and limitation of motion of his neck, he was not aware of any other impairment of function. He did not seek medical aid but returned for examination in response to a letter written to him in preparation for this paper. Roentgenograms taken in the lateral projection showed the fracture of the odontoid process still to be nonunited and there was 6 to 7 mm. of mobility on flexion and extension of his neck (Fig. 4). No further treatment has been given to him.

ANALYSIS OF SURGICALLY TREATED CASES

DECOMPRESSIVE LAMINECTOMY

(1) *#114489.* This 21-month-old child was discussed previously under "Deaths" (1). The dislocation of the atlas was not recognized with certainty before surgery. Skeletal traction would have been the preferred treatment, followed later by fusion if the child had survived.

FUSION OF OCCIPUT TO C3

(1) *#167647.* This patient, also previously referred to under "Deaths" (2), had metastatic carcinoma of the prostate to the atlas with dislocation of C1 on C2. Wiring of fragments of bone from the occipital bone to C3 successfully held the dislocation in the position of satisfactory reduction already attained by skeletal traction. The patient died of pulmonary embolus on the 14th postoperative day. At

necropsy the operative fixation was found to be solid by virtue of firm wiring of fragments of bone. Though perhaps effective, this type of fusion is unduly arduous and no more stable than fixation of the atlas to the axis.

FUSION OF ATLAS TO AXIS

Two patients were treated in 1949 and 1950 by surgical fusion between the axis and atlas only:

(1) *#113411.* A 28-year-old woman was first seen 3 weeks after an automobile accident in another city. There was no neurologic disability. She had extreme pain in her neck and mobility of the neck was restricted. The dislocation was reduced satisfactorily by application of skeletal traction, after which fusion was performed of C1 to C2 by ilial grafts wired to the laminae of C1 and C2. There was definite forward displacement of the atlas on the axis 3 months after operation. Although her symptoms were considerably improved, this method of fusion was not considered satisfactory. She was seen last in March, 1956, 5 years and 9 months after operation, when she had some soreness in her neck, limitation of movement of the neck of about 15 per cent, but no neurologic disability. The range of motion in the fusion in flexion and extension of the neck was about 1 mm., but there was still slight anterior dislocation of C1 on C2.

(2) *#93205.* A 67-year-old man walked into the emergency room following a fall down a flight of stairs while under the influence of intoxicants. The posterior dislocation of the atlas on the axis was so pronounced on the first lateral films of the cervical spine (Fig. 5, left) that it was amazing to those in attendance that he showed no neurologic disability. Immediate complete reduction of the dislocation was obtained by the use of skeletal traction, and 8 days later a fusion was performed of the laminae and spinous processes of C1 and C2 with wiring of the fragments of bone. Because of his age he was gotten out of bed almost immediately with traction applied by rubber straps between the Crutchfield tongs and an external metal support attached to a plaster jacket. There was no further follow-up until he returned in response to a letter during the preparation of this paper. At the age of 73 years he showed no neurologic disability. The movements of his neck were somewhat limited but not more than commonly seen in a person of his age. Roentgenograms showed slight posterior dislocation of 2 to 3 mm. of the atlas on the axis with apparent healing of the fractured odontoid process (Fig. 5, right).

FUSION OF C1, C2, C3

The operative method of fusion using iliac bone for grafting, and, more recently, using ribs from a bone bank, was used in 12 cases. The operations were performed from 8 days to 3 months after injury.

Since roentgenograms are of such vital importance in reaching a diagnosis of fracture of the odontoid process, some attention should be devoted here to the technique of taking the films. The conscious or unconscious patient who is suspected of having an injury of the cervical spine—including all serious injuries of the head—is not moved to the diagnostic roentgenographic table until a horizontal-beam lateral film of the cervical spine is taken to rule out a fracture-dislocation. If a high cervical lesion is indicated, either clinically or in the initial films, a special small cone is used to see the C1–C3

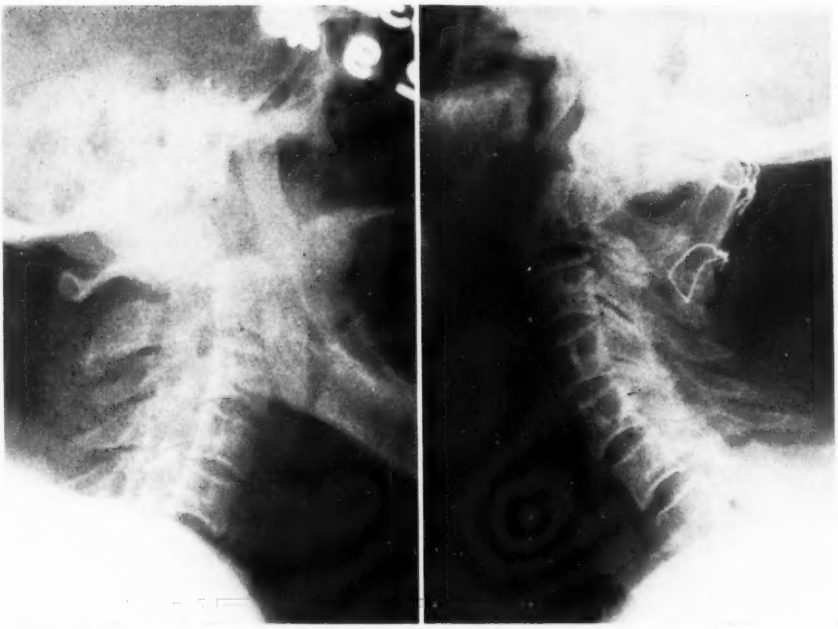
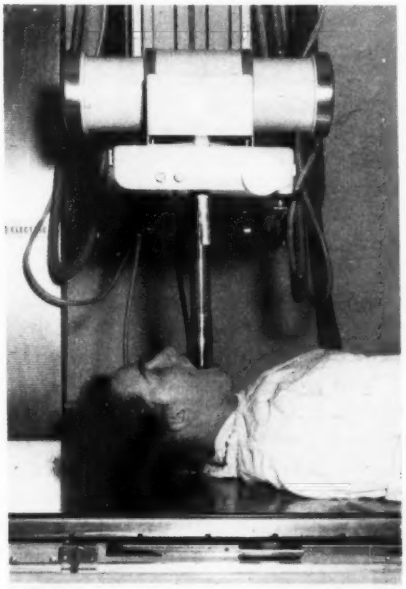


FIG. 5. Man, aged 67 years, with no neurologic deficit. (*Left*) Posterior dislocation of C1 on C2. This was reduced by traction. (*Right*) Film 6 years later shows solid fusion.

FIG. 6. Special small cone for obtaining anteroposterior views of odontoid process through open mouth.



area. A cone small enough to be inserted into the mouth may be used in order to obtain a clear anteroposterior view of the odontoid process (Fig. 6).

OPERATIVE TECHNIQUE

Crutchfield tongs are inserted as soon as a diagnosis of fracture of the cervical spine is made. Enough traction (up to 20 or 25 pounds for a few minutes) is used to reduce the dislocation. After reduction is accomplished, the weight is reduced to about 5 to 10 pounds, and the position of the dislocation is checked by lateral-view roentgenograms.

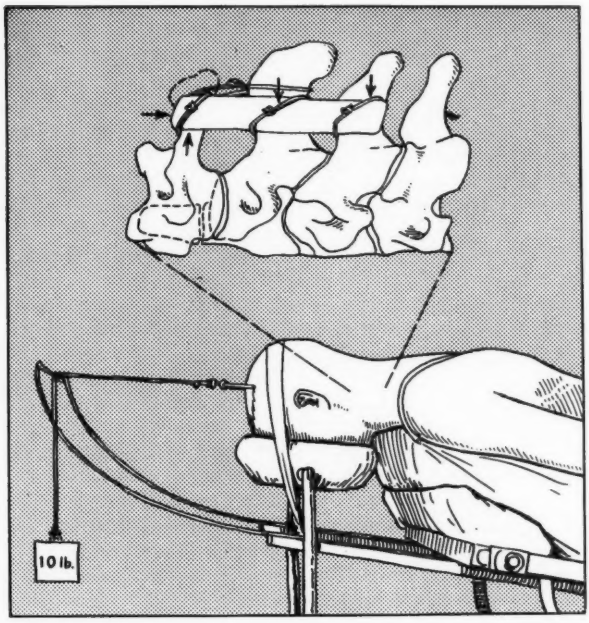


FIG. 7. Artist's conception of patient placed in prone position in the cerebellar head-rest without flexion or extension of the neck. Insert shows fusion from lateral view.

As soon as the general condition of the patient permits it, operation is done under general anesthesia. An endotracheal tube is inserted blindly by the intranasal route, with the patient's head in traction and without dorsiflexion of his neck. He is then placed in the prone position in the cerebellar head-rest without flexion or extension of the neck (Fig. 7). About 10 pounds' traction is maintained during the exposure which is accomplished by sharp dissection, clearly exposing the occipital bone, foramen magnum, arches of the atlas and axis, and the 3rd and 4th cervical vertebrae. Exposure is carried out to the facets on both sides.

If the procedure is performed within 10 days of the injury, discoloration usually will be seen of the deep muscles of the neck and occasionally also of the dorsal surface of the dura mater. Later there may be no abnormality evident in the exposed field except for unusual mobility of the axis.

All ligamentous attachments to C1, C2, and C3 out to the facets are removed with rongeurs. The cortex of the laminae of C1, C2, and C3 is removed by multiple small bites with a rongeur until medullary bone is exposed. A small motor-driven burr would be ideal but is considered hazardous. Care must be taken at this stage of the procedure to avoid breaking through the thin laminae of C1 and C3, but C2 is usually thick enough for the cortex to be removed easily. In order to facilitate the passage of wires

beneath the laminae, they are made narrower in the cephalad-caudad dimensions by removing small portions of the upper and lower edges with rongeurs.

Stainless-steel wires, #20 gauge, are passed beneath the laminae of C1, C2, and C3, using a curved needle large enough to accommodate the wire. The heavy wire is bent sharply as it passes through the eye of the needle in order to reduce the bulk of the needle and wire passing between the lamina and dura mater. Occasionally the wire may be passed without a needle but, of course, caution must be taken not to penetrate or encroach on the dura mater which is left intact throughout the procedure. Two wires are passed laterally under the right and left laminae of C1, C2, and C3, and another strand of wire is passed beneath the midportion of the atlas. This wire is looped around the heavy spinous process of the axis. It is tied after traction is reduced to 5 pounds and, if necessary, the neck is extended slightly.

Ribs obtained from sterile thoracic procedures and preserved in the bone bank are used as grafting material, or an iliac graft may be taken from the patient at the time of surgery. The short fragment of rib to be used is cut to the appropriate length to extend from the cephalad edge of the lamina of the atlas to the caudad edge of the lamina of the 3rd cervical vertebra (Fig. 8). The side of the rib opposite to the underlying laminae is prepared by removing the cortex of the rib to fit the similarly denuded laminae. The rib on each side is then tied in place with three wires. At times additional

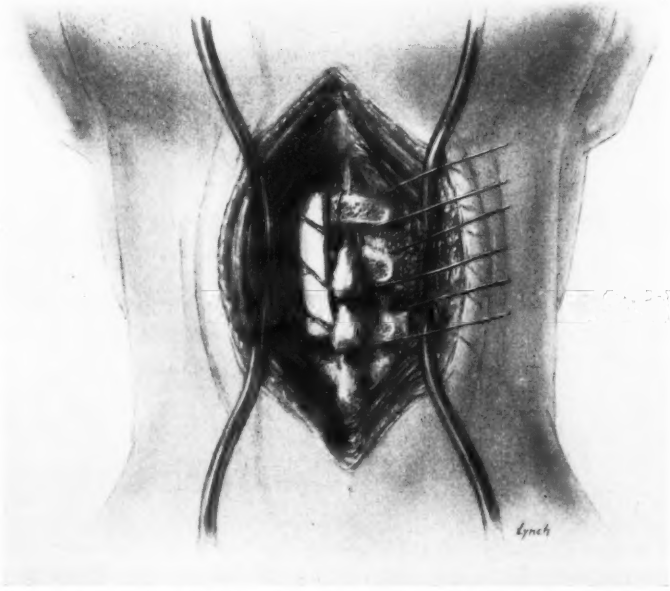


FIG. 8. Artist's conception of graft using short fragment of rib extending from cephalad edge of the lamina of atlas to caudad edge of lamina of 3rd cervical vertebra.

chips of bone are placed in the wound in contact with the denuded laminae and over the exposed facets, but this is not considered essential. The wound is closed in multiple layers with interrupted sutures of silk.

DISCUSSION

Of the 12 operations performed by fusing C1, C2, and C3 with bone graft, 1 was done over 6 years ago, 2 over 2 years ago, 5 over a year ago, and the other 4 from 3 to 12 months ago.

Only 3 of these patients showed any neurologic disability from the injury of the odontoid process:

One of these, on whom operation was performed over 6 years ago, sustained a severe injury of his head together with multiple contusions and abrasions. He had multiple subcutaneous abscesses, and it was difficult to maintain his neck in proper traction because of his restlessness. He became totally tetraplegic, but in spite of the known abscesses elsewhere in his body, fusion of C1, C2, and C3 was performed. Except for one superficial abscess at the site of a suture, the wound healed by primary intention. He made a steady recovery and was last examined 1 year postoperatively. There was slight hyperactivity of the deep tendon reflexes on the left side as compared with the right, the motions of the neck were limited about 20 per cent, and he was going to school regularly. Roentgenograms showed his neck to be in normal position with solid healing of the grafts of bone (Fig. 9).

A second patient had some weakness of the left arm and leg as a direct result of the injury. The weakness improved before surgery was performed but was accentuated by the surgical procedure for 5 days postoperatively. After operation he continued to improve and 8 months later his neurologic findings were normal.

Although there was considerable neurologic disability immediately after injury, a third patient recovered completely and returned to his occupation of teaching school.

Three other patients of the group presented specific problems:

A 22-year-old man was thrown from an automobile in an accident. He showed no neurologic abnormality but roentgenograms disclosed a fracture of the odontoid process, backward dislocation of the atlas on the axis, and marked lateral dislocation of the atlas on the axis to the right. Crutchfield tongs were inserted and the dislocation was reduced by application of 30 pounds of skeletal traction without manipulation. He was transported in skeletal traction to the North Carolina Baptist Hospital where cervical fusion was performed on the 8th day after injury (Fig. 10). Four months after operation he was doing light work, wearing a Thomas brace on his neck. Eight months later he returned to work with no pain of his neck. There was about 15 per cent limitation of rotation and lateral bending of his neck.

A 63-year-old Negro man was injured when thrown against a barn by a cow. Some days later satisfactory roentgenograms in the lateral and anterior position showed anterior dislocation of the atlas on the axis without fracture of the odontoid process. The anteroposterior films were taken through the open mouth. Fusion was performed 1 month after injury, the wound healed satisfactorily, and the patient

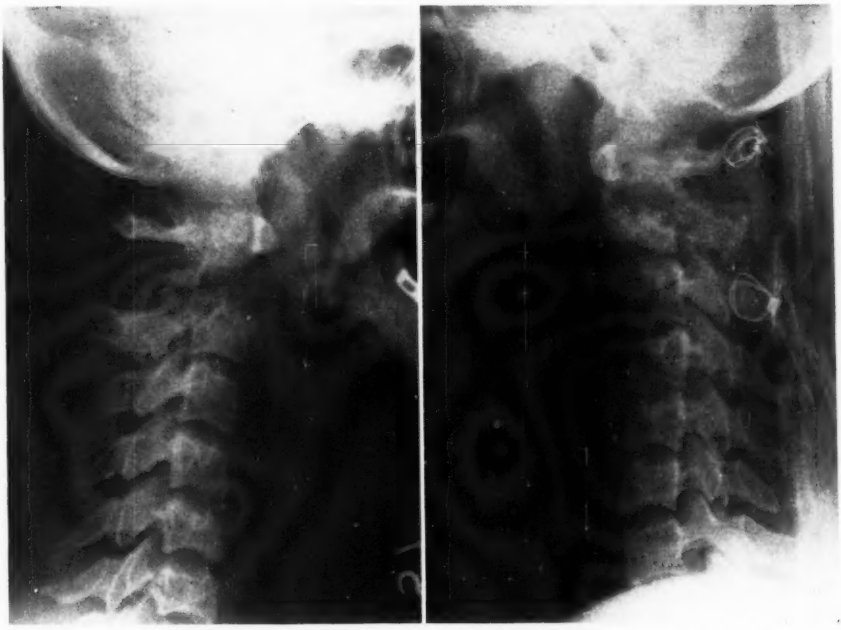


FIG. 9. Fracture-dislocation of odontoid process associated with severe injury of head and extreme restlessness. Separation of fragments by traction was necessary to overcome progressive tetraplegia. Film on right, 41 days postoperatively, shows beginning healing of fracture. Patient made almost complete neurologic recovery following fusion.

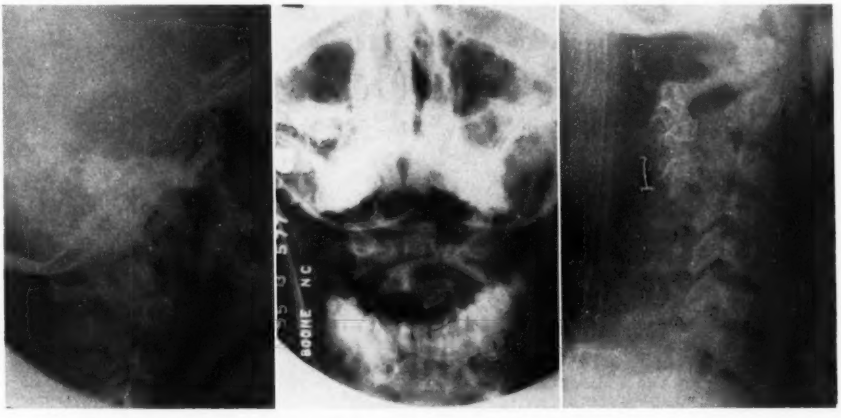


FIG. 10. Films on left and in center show posterior and lateral dislocation of C1 on C2 with fracture of odontoid process. No neurologic deficit. Dislocation readily reduced by skeletal traction with 30 pounds of weight for 30 minutes. On right, immediate postoperative fusion.

was fitted with a Thomas brace on his neck at the time of discharge. This case, covered by a Compensation Commission, was punctuated by considerable unpleasantness between employee and employer, and it has been difficult to determine whether or not his continued complaint of pain in his neck is valid. Roentgenograms 10 months later showed the atlas and axis maintained in the same position as fused, but anteroposterior films showed a clear line of decreased density across the base of the odontoid process, probably representing a fracture. There was no displacement of the odontoid process from the axis (Fig. 11).

A 47-year-old man with a past history of severe rheumatoid arthritis several weeks before admission began to have increased pain in the neck and increasing clumsiness of the arms. A roentgenogram showed a forward dislocation of the atlas on the axis without fracture of the odontoid process. This was reduced easily by application of traction and subsequently fusion of C1, C2, and C3 was carried out. The function of his arms improved postoperatively.

Of the 3 patients who had rupture of the transverse ligament, in 2 it was caused by trauma and in the third it was associated with severe rheumatoid arthritis. Although the forward dislocation of the atlas on the axis was marked in each of the 2 patients who had been injured, there was no movement on flexion and extension of the neck. Furthermore, at operation in each case the atlas was firmly fixed in its forward position and could not be moved by gentle manipulation with rongeurs as in the other patients with fracture of the odontoid process. In the patient with rheumatoid arthritis there was free mobility between the atlas and axis, and complete reduction was accomplished by traction before surgical fusion was done.

In all but 1 of the patients on whom fusion of C1, C2, and C3 was done, the grafts of bone were clearly visible and solid as long as over 6 years after operation. In the 2 patients in whom only C1 and C2 were fused, slight mobility of the atlas on the axis was revealed on films taken in flexion and extension of the neck, but there was no detectable movement in patients with fusion of C1, C2, and C3. Most of the patients were free of significant pain or discomfort within 30 days after fusion, although the rotary movement of the cervical spine was limited 15 to 30 per cent. Some patients returned to light work wearing a brace 2 months after operation (Fig. 12).

One man with a fractured odontoid process treated by fusion of C1, C2, and C3 had a rather severe episode of pain in his neck with limitation of all movements of the neck about 18 months after surgery was done. Roentgenograms repeated then showed the grafts of bone and wires in place, and no cause was found for his pain, from which he recovered spontaneously.

SUMMARY

1. Fractures of the odontoid process carry a high mortality if associated with acute injury to the cervical spinal cord.

2. The immediate mortality is low if there is no severe injury to the cervical spinal cord—that is, if the patient reaches the hospital in satisfactory neurologic condition.

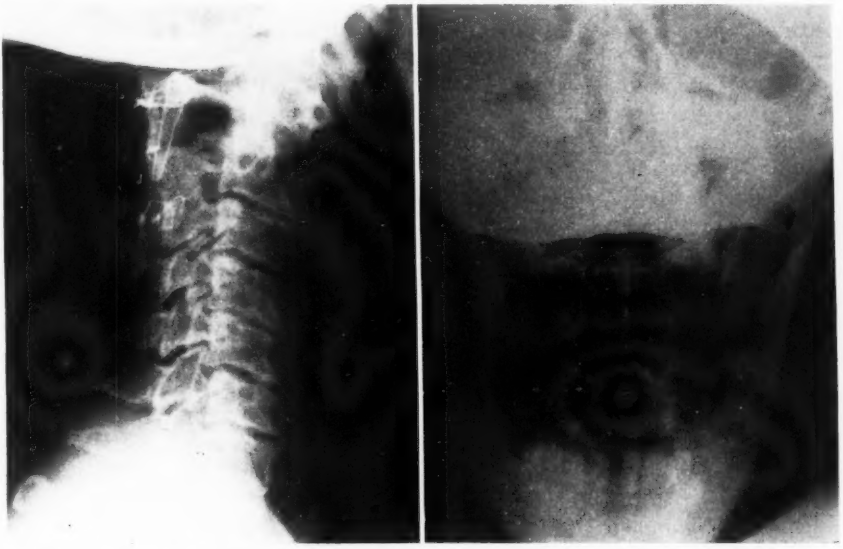


FIG. 11. Man, aged 63 years. (*Left*) Postoperative fusion of C1, C2, and C3. Forward dislocation of C1 on C2 brought about by rupture of transverse ligament. Preoperative films disclosed no fracture of odontoid process in the anteroposterior or lateral views. (*Right*) Anteroposterior film 1 year after operation shows fracture of base of odontoid process. Fusion solid.

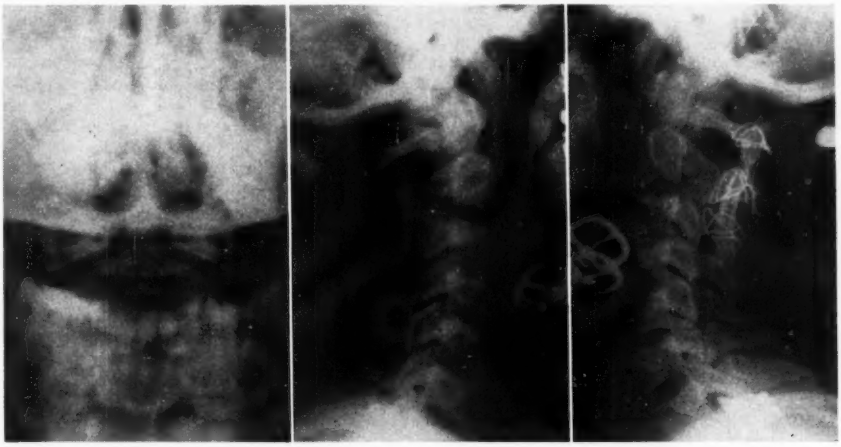


FIG. 12. Roentgenograms in anteroposterior and lateral projections showing fracture through base of odontoid process without significant displacement. On right, 1 month after fusion, patient was ambulatory, wearing adjustable Thomas collar.

3. With prolonged immobilization of the head and neck, some fractures of the odontoid process do undergo solid healing apparently, but the period of disability is lengthened.

4. Late neurologic complications with eventual death up to 30 years after unsatisfactory healing of a fractured odontoid process have been reported frequently in the past and are serious enough to warrant operative fusion to prevent future dislocation of the atlas on the axis.

5. The lessons learned from experience with 25 fractures of the odontoid process are reviewed.

6. Operative fusion was done on 15 patients with only 1 death from pulmonary embolus on the 14th postoperative day of a patient with metastatic carcinoma of the prostate.

7. A detailed technique of performing cervical fusion is discussed and the method of fusion of C1, C2, and C3 is described. This proved satisfactory in 12 patients seen over a number of years.

8. Experience gained from this relatively large group of surgically treated patients, all carefully followed postoperatively, leads to the conclusion that early surgical fusion in cases of fracture of the odontoid process protects the patient from the late sequelae of atlanto-axial dislocation. Fusion can be done with much shorter hospitalization and earlier return to useful employment than with prolonged traction and immobilization which does not assure solid healing of the fractured odontoid process.

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CAROTID ARTERY COMPRESSION AS EMPLOYED BOTH IN THE PAST AND IN THE PRESENT

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(Received for publication June 10, 1957)

THE term carotid is derived from a Greek word (κάρος) meaning sleep or stupor, suggesting a function for an artery which has been known for many centuries. Most of the observations made in antiquity have resulted from ligation of the artery rather than from temporary compression. At the time of Galen[13] it was believed that the effect from compression was caused by "compression of the sensitive nerves which lie near the arteries." The Persian, Avicenna[4] (980–1037), stated that when the carotid arteries were ligated sense and motion were lost. If the carotid arteries were temporarily obstructed, Valverdus[30] noted early that we immediately grow stupid and fall asleep—"he saw this experiment made by Columbus at Pisa in the year 1554, on a young man, in a large circle of gentlemen, equally to their terror and to the amusement of the operators, who persuaded them that it was done by the force of incantation."

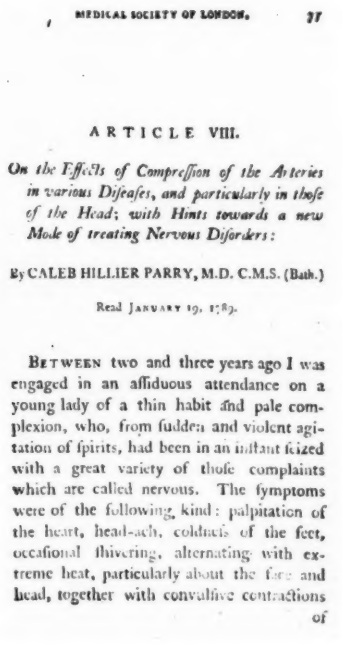
Many experiments through past centuries were then made upon ligation of the carotid arteries in several species of animals. The results were contradictory and confusing. In 1845 Norman Chevers[5] reviewed the history of the effects of carotid ligation in animals and man up to that period. Clinical observations were correlated with pathological findings and his conclusions conformed essentially with modern views. He referred to carotid compression only in a brief footnote. Chevers stated that Dr. Caleb Parry[26] practised compression of the carotid arteries in patients having mania, headache, vertigo, convulsions, and hysterical complaints. Others also used this method, including a Dr. Allier[1] in 1837 who believed it to be efficacious in some cases of epilepsy. Allier had compressed both carotid arteries simultaneously in a case of hydrophobia at the beginning of a convulsive paroxysm. "In an instant the convulsions ceased, and the patient fell into a kind of fainting fit. Alarmed at this, the family refused to allow the doctor the methodical employment of this powerful treatment." Chevers pointed out that regardless of the therapeutic use such compression was put to, it proved that "obstruction of these vessels has a powerful effect upon the brain, and that in compressing the arteries, there is not produced sufficient impediment in the internal jugular veins to occasion dangerous cerebral congestion."

Hart[17] stated that Professor T. Vanzetti[32] of Padua claimed priority as inventor of digital compression. Vanzetti insisted upon the value of digital

* Aided by the Kresge Foundation.

compression; studied its application to the treatment of aneurism and thus claimed the attention of European surgeons. "In July 1856 a female patient came under the care of Professor Gioppi, of Padua, the subject of an aneurism of the ophthalmic artery . . . The pulsating aneurismal tumor could be felt by introducing the finger between the globe of the eye and the roof of the orbit; the bruit could be heard loudly. The carotid was compressed for periods of a minute or two, and, with frequent intervals, by the convalescents of the ward and by the patient herself. Pressure continued for more than a minute produced fainting. Nevertheless, at the end of one day there

Fig. 1. Caleb Hillier Parry in 1789 reported upon his experiences in using digital compression of the carotid artery as a method of treatment. He noted that syncope resulted from compression.



MEDICAL SOCIETY OF LONDON. 37

ARTICLE VIII.

On the Effects of Compreſſion of the Arteries in various Diſeaſes, and particularly in thoſe of the Head; with Hints towards a new Mode of treating Nervous Diſorders:

By CALEB HILLIER PARRY, M.D. C.M.S. (Bath.)

Read JANUARY 19, 1789.

BETWEEN two and three years ago I was engaged in an aſſiduous attendance on a young lady of a thin habit and pale complexion, who, from ſudden and violent agitation of ſpirits, had been in an inſtant ſeized with a great variety of thoſe complaints which are called nervous. The ſymptoms were of the following kind: palpitation of the heart, head-ach, coldneſs of the feet, occaſional ſhivering, alternating with extreme heat, particularly about the face and head, together with convulſive contractions of

Fig. 2. Title page of Parry's paper in the Memoirs of the Medical Society of London.[26]

was a visible improvement, and at the end of four days all pulsations had ceased. Four months afterwards the eye rested and moved naturally in the orbit."[17] A second case of aneurism of the ophthalmic artery, cured by digital compression, was published in 1858 by Vanzetti. "Intermittent compression of the carotid by the finger was here also employed for five minutes at a time, and cure was effected by seven hours and twenty minutes of compression spread over eighteen days."[17]

However, an earlier clinical use of carotid compression was described with case reports in 1789 by Parry in a paper read to the Medical Society of London[26] (Figs. 1 and 2). Compression of the carotid artery was to reduce "the too great quantity of blood forced through the carotid arteries into the

brain" and was accomplished by pressing "strongly with my thumb on the carotid artery a little below the larynx." Parry reported that since the period of this discovery for more than a year and a half he had made the experiment in a great variety of instances with success in hemicrania, nervous headache, vertigo, noises in the head, in the mental agitation of nervous patients, hysterical states and in aborting epileptic attacks. In the latter instance the patient applied the compression when warned of an impending convulsion. In palpitation of the heart (motus cordis vehemens abnormis) with "undue determination"of blood to the brain, associated with various symptoms, the method was particularly effective. In the use of compression, which he rarely "continued more than a minute or two," he found that it induced "almost immediately a sound sleep" and also "a kind of sickness like that which accompanies the beginning of syncope." It often produced "a great deal of anxiety about the heart which the patient attempts to relieve by usually deep inspirations of sighing."

Magendie[22] in 1838 described the rise in heart rate and blood pressure which followed upon obstructing the carotids. He and others, according to Ask-Upmark,[3] ascribed the effect to the altered blood supply to brain centers.

Kussmaul and Tenner[21] in 1859 contributed a complete review of the subject (Figs. 3 and 4). These authors stated that during the 19th century compression of the carotid was introduced as a remedy by Parry and brought into wide use. No mention was made of Vanzetti. Effects of carotid compression, they stated, were also studied by Jacobi[20] of Siegburg who performed some hundreds of experiments with results confirmed by other clinicians of that period. Jacobi had noted the following symptoms as generally observed: "dimness of sight, dizziness, stupor, weakness in the legs, staggering, swooning, loss of consciousness, and sudden apoplectic falling-down."

Kussmaul and Tenner did six experiments in adults in whom complete compression of both carotid arteries was made, and concluded that this "may occasion loss of consciousness, dilatation of the pupils, prolonged breathing, and generalized convulsions,—in short, all the symptoms of a slight epileptic attack." Others had not reported these convulsive moveents attendant upon carotid compression.

Waller[33] in 1862 and Czermak[9] in 1866 found that cardiac slowing occurred when the upper portion of the carotid arteries was compressed. Both related the effect to stimulation of the vagus nerve (as did Galen). Ask-Upmark[3] stated that for more than 50 years the interpretation given by Czermak was generally accepted and the test was termed "Vagusdruckersuch."

Naunyn,[24] who succeeded Kussmaul at Strassburg, experimented upon his pupils and found that in 6 (about 30 years of age) compression resulted in no effect or slight dizziness. In 2 (over 50) after a few seconds' pressure there was loss of consciousness with slowing of the pulse and a slight convulsion. On pressure being removed there was immediate return to consciousness with no further disturbances. When epileptiform attacks occurred there was al-

ways arteriosclerosis and this observer believed that the effects of the pressure were caused by impairment of blood flow to the brain which did not get enough from the vertebral arteries and that the circle of Willis was damaged (Crile[7]).

Concato[6] (1870) noted also that in young people compression produced no effects but in patients with arteriosclerosis, epileptiform attacks resulted. He recommended that pressure be applied over the carotid bulb. An interesting result of the test was reported by de la Harpe,[16] "where pressure on the carotid caused intermittency of the pulse. Pressure on the right carotid pro-

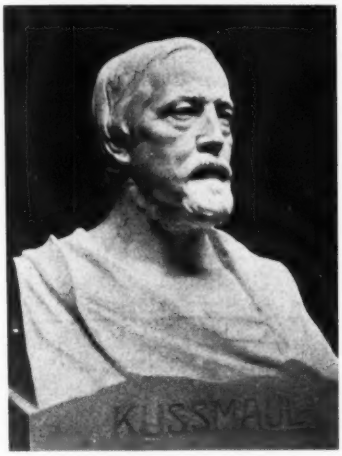
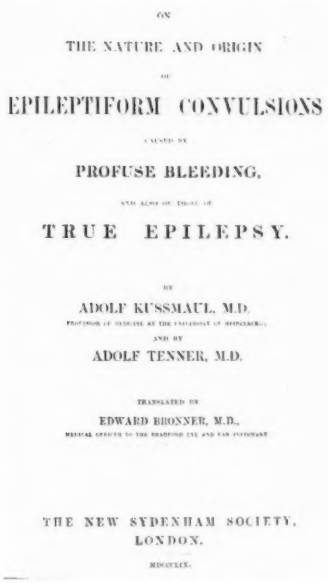


FIG. 3. Adolf Kussmaul and Adolf Tenner first reported that generalized convulsive movements attended the syncopal state induced by digital compression of both carotid arteries.


ON

THE NATURE AND ORIGIN

OF

EPILEPTIFORM CONVULSIONS

CAUSED BY

PROFUSE BLEEDING,

AND ALSO OF THAT OF

TRUE EPILEPSY.

BY

ADOLF KUSSMAUL, M.D.

AND BY

ADOLF TENNER, M.D.

TRANSLATED BY

EDWARD BRONNER, M.D.,

THE NEW SYDENHAM SOCIETY,
LONDON.

MDCCCLIX.

FIG. 4. Title page of Kussmaul and Tenner's paper dealing with convulsive movements related to ischemia of the brain, published in 1859.[21] This work was overlooked by most authors who had investigated the reflex mechanism of the carotid sinus.

duced more marked effect than on the left, and slight pressure was equally effective as deep pressure. Ordinarily the pulse would not drop more than ten to fifteen beats per minute, . . . Once it dropped from 96 to 48" (Crile[7]). The author attributed this to a "slight excitation" of the vagus nerve caused in turn by an "exaggerated impressionability of the heart muscle." He could not explain the difference of effect following pressure on the right and left carotid. (The patient, aged 65, who showed this response had just had an attack of apoplexy.)

Crile[7] (1901) reported experiments in both dogs and humans in which both carotid arteries were safely, temporarily occluded by clamping for various periods of time (in animals for from 24 to 48 hours). In 9 patients varying in

age from 58 years to 7 months, both arteries were clamped in carrying out radical excisions of structures of the head and neck. All recovered but one, who died from hemorrhage on the 13th day. The patients were placed in the Trendelenburg position for the operation (the time for each procedure was not stated).

In 1900 Siciliano[29] stated that an increase in blood pressure and pulse rate upon clamping the common carotids occurred too rapidly to be the result of cerebral anemia, thus disputing the older views (Magendie). He found also that if the internal carotids were clamped no rise in blood pressure resulted. He concluded that a reflex was responsible related to a reduction of pressure within the carotid.

Sollmann and Brown[30] (1912) found that the blood pressure fell upon pulling the common carotid heartwards. No effect occurred when it was pulled headwards or when pulls of the vagus or sympathetics were made.

Hering[18] (1927) reported that the carotid sinus was associated with a neural mechanism involved in the regulation of the cardiac rate and the arterial blood pressure. When the carotid sinus was compressed at its bifurcation, thus increasing the intravascular pressure at this point, there occurred a slowing of the heart, vasodilatation and a fall in blood pressure. When the common carotid artery was compressed below the sinus area, the intravascular pressure within it was reduced and then cardiac acceleration, vasoconstriction and a rise in arterial pressure occurred. These effects were stated to result from afferent impulses mediated from nerve endings located on the carotid sinus through the carotid sinus and the glossopharyngeal nerve to vagal nuclei in the medulla.

Heymans[19] and his associates added laboratory findings to support the concept that the carotid sinus reflexes function as a sensitive pacemaker of the circulation (Weiss[37]). The nerve endings of the carotid sinus were found to be of two types, pressoreceptor and chemoreceptor components of a reflex mechanism mediated mainly through the glossopharyngeal nerve.

It was noted early that the effectiveness of these reflexes on various parts of the cardiovascular system varies in different species of animals depending in part on the role of the vagus and sympathetic nerves. In man considerable variations exist in both health and disease (Weiss[37]).

Weiss[37] and Weiss and Baker[38] developed the clinical application of the reflex and suggested three types of carotid sinus responses: "A vagal type" (with cardiac asystole) in which atropine and epinephrine prevent the effect. "A depressor type" (with a fall in arterial pressure); atropine has no effect but epinephrine aborts the attacks. "A cerebral type" (without cardiac asystole or hypotension) unaffected by atropine or epinephrine. The total flow of blood through the brain (in the cerebral type) in contrast to the other types is normal during the stage of unconsciousness and convulsions.[38] After many classes of medical students were familiarized with the streetcar conductor and his celluloid collar inducing syncope upon turning of the head, both the carotid sinus reflex as a clinical entity and the collar were de-empha-

sized. Gradually also the importance of pressor receptors in the carotid bulb lessened as newer information was gained in the laboratory (Schmidt[28]).

In 1943 the effects of acute cerebral anoxia in man were studied by a technic employing a Kabat-Rossen-Anderson[27] cervical pressure cuff devised to arrest acutely the cerebral circulation. Studies were made in 11 schizophrenic patients and 126 normal young males. "The characteristic reactions resulting from acute arrest of the circulation in the brain for five to ten seconds were fixation of the eyeballs, blurring of vision, constriction of the visual fields, loss of consciousness and anoxic convulsions." Delta waves appeared in the electroencephalographic records at the point of loss of consciousness. The recovery was quick and uneventful; the procedure was stated to be free from danger.

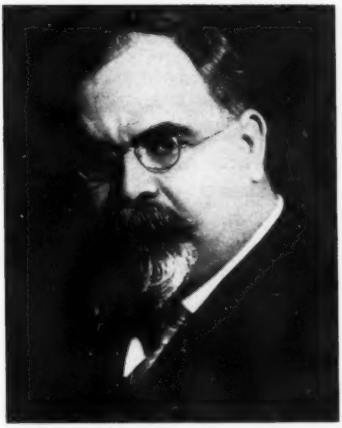


FIG. 5. Rudolph Matas emphasized in his reports the importance of increasing collateral circulation by temporary compression of an artery before ligation, including the carotid artery (the Matas test).

With the advent of angiography, a revival of carotid compression occurred mainly as a preoperative test to determine the patient's tolerance for ligation of the carotid artery in the treatment of saccular aneurisms of the intracranial portion of the internal carotid and carotid-cavernous fistulae. This maneuver had been recommended early by Matas[23] (Fig. 5) who stated that collateral circulation was developed or enhanced by such compression as patients developed a tolerance to the compression. Halsted[15] and Dandy[10,11] also believed such compression before ligation was worth while. Evidence to indicate the validity of these views has recently been submitted.[36]

Interest was taken in noting the electroencephalographic records of patients upon whom carotid compression was applied and evidence of cerebral ischemia was occasionally recorded, especially when compression was not well tolerated.[2] Reports of electroencephalographic responses among patients with a sensitive carotid sinus were also made by various authors.

There are many contemporary reports concerned with "stimulation" of the carotid sinus but none devoted to carotid compression as a diagnostic aid in evaluating occlusive cerebral vascular disease. Denny-Brown[12] in 1952 stated that in internal carotid thrombosis the reaction to manual compression of the internal carotid artery on the side of the symptoms will reproduce the symptoms within 30 seconds in about half the cases and is a valuable confirmatory sign. In 1954 Ochs *et al.*[25] reported 7 cases of thrombosis of the internal carotid artery in which studies were made of the cerebral circulation. They stated that "The response to unilateral compression of the com-

mon carotid artery below its bifurcation and the carotid sinus is a useful procedure in the clinical recognition of primary thrombosis of the internal carotid artery. The induction of syncope by this maneuver may be considered as strong presumptive evidence of occlusion of the contralateral internal carotid artery." In 2 of the 7 cases reported this test was found to be of diagnostic value. They pointed out that "the carotid sinus mechanism is not the only and probably not the most important factor involved in the causation of syncope when it occurs in subjects with this disorder." However, syncope, in general, when induced by "stimulation" or "compression" continues to be interpreted only in terms of a carotid sinus reflex or an anatomical abnormality of the circle of Willis.

Our interest in the responses to carotid compression followed its employment first in patients with thrombosis of the carotid artery having a recent hemiparesis or hemiplegia.[35] An old practice was thus related to a rather common disease as disclosed by the new technic of angiography. When the contralateral functioning artery was compressed the effect was often dramatic. Syncope associated with convulsive movements usually occurred in 10–20 seconds. This was at first ascribed to a sensitive carotid sinus reflex suggested by Weiss[37] as the "cerebral type." Further study of the response by electrocardiographic, electroencephalographic and blood pressure records, however, indicated that the cerebral blood pressure fell and the cerebral blood flow was not adequate to meet the requirements to maintain consciousness. Convulsive movements, pallor, increased respirations, dizziness, and blurred vision were other evidences of the temporarily induced cerebral ischemia. Some patients were able to tolerate the compression when the carotid thrombosis was of long duration (months), having established collaterals, but in cases of recent thrombosis they usually did not.

When compression was used as a routine diagnostic clinical test prior to angiographic study in patients suspected of a complete carotid occlusion it was found that a partial occlusion also was frequently accompanied by syncope upon contralateral compression.[35] Angiographic study of the carotid bulb area often visualized the localized atheromatous abnormality. Only minor changes occurred in the blood pressure and pulse. It was found to be important that both the external and internal carotids be compressed totally to produce a maximal effect and thus a satisfactory test. The first compression test usually gave the greatest response. Thereafter, patients seemed to develop tolerance to the compression. In view of this, compression has been and is being carried out daily in patients presenting the neurologic findings of partial occlusive disease of the carotid with cerebrovascular insufficiency. The patients compress their carotids to the point of producing mild dizziness. Gradually tolerance develops and in some there is also an improvement in symptoms.

A few patients with occlusion of the vertebral artery—complete or partial—have been encountered. Both right and left carotid compression re-

sulted in syncope or near syncope, again if the lesion was of recent occurrence.

An unexpected response to testing by compression was noted in patients demonstrated by angiography as having an occlusion of one anterior cerebral artery.[14] These patients were of two classes: those with neurophychiatric symptoms and those with added hemiparesis or hemiplegia. The neuropsychiatric symptoms were commonly bizarre including anxiety, generalized weakness, spells of weakness, dizziness, fears, depression, sense of "passing out," nervousness, distraughtness, and disorientation. The neurological findings were often normal. The role of the anterior cerebral artery may have a neurological significance beyond that commonly acknowledged. Not only may its function be related to the maintenance of consciousness but dysfunction of the artery may underlie neurologic and psychiatric disturbances undisclosed by previous studies[8] which did not evaluate the presence of occlusive disease of the carotid arteries in the few cases upon which the paralytic syndrome of the anterior cerebral artery has been established.

A distinction between the role of the anterior cerebral artery and that of the middle cerebral artery as related to consciousness is indicated in the absence of a syncopal response upon compression of the contralateral carotid in patients with occlusion of the trunk of the middle cerebral artery. Also with few exceptions (carotid sinus reflex or partial carotid occlusion) patients presenting a hemiplegia with normal-appearing angiographic patterns do not develop syncope.[34] Arteriosclerosis of small arteries involving the terminal distribution of the middle cerebral artery may account for this response.

In an analysis of 100 patients presenting a hemiplegic or hemiparetic neurologic state, compression tests were performed in conjunction with routine neurological studies including angiography (Fig. 6). The responses are shown in Table 1, with illustrative angiographic pattern. Exceptions or misleading responses were found to occur in 15 per cent of the patients. These were noted mainly in elderly patients with occlusive disease at several points in the cerebrovascular system, patients with a "sensitive carotid sinus" or those with increased intracranial pressure.

SUMMARY

This paper has summarized the uses of and the responses to digital compression of the carotid artery in humans during the past centuries. The earliest observers related the unconsciousness resulting from compression to the vagus nerve. Caleb Parry in 1789 employed carotid compression to reduce blood flow to the brain as a treatment and found that unconsciousness occurred and that palpitations of the heart improved. Later in the next century (1859) Kussmaul and Tenner noted that besides unconsciousness, convulsive movements may accompany ischemia of the brain from carotid compression when induced bilaterally. During the present century Hering (1927) and

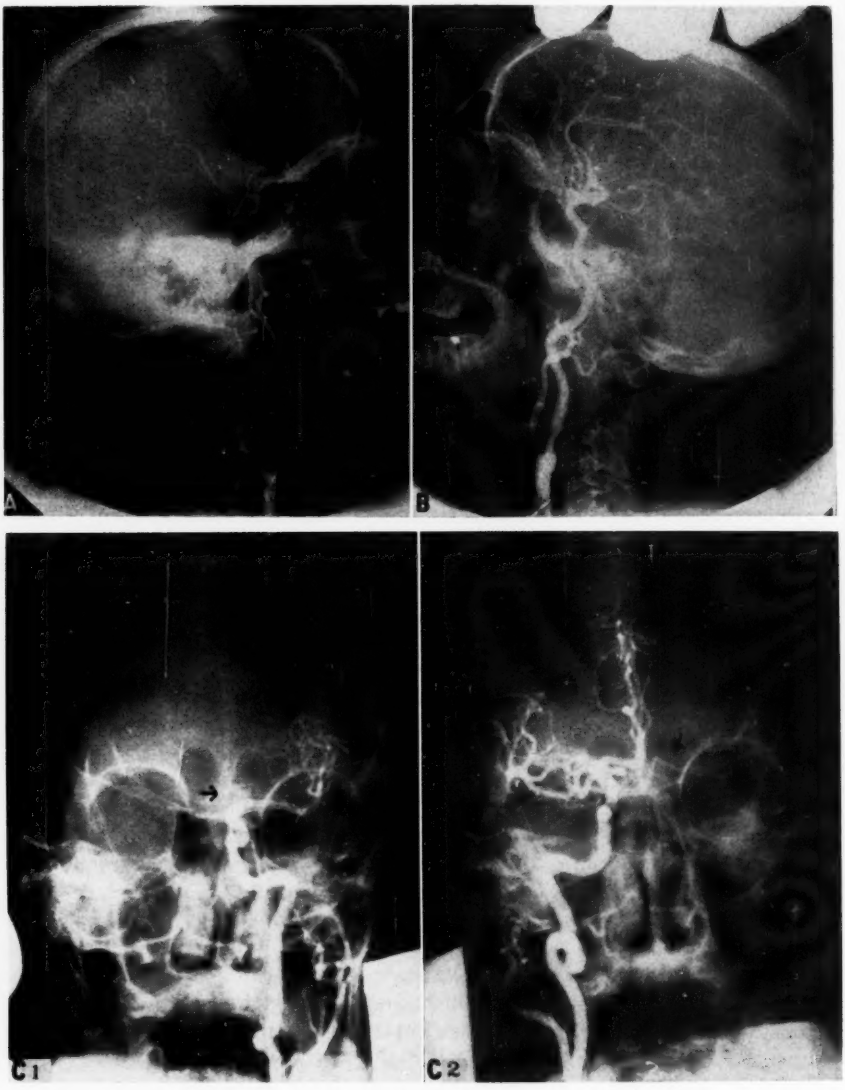


FIG. 6. Angiograms showing (A) total carotid occlusion; (B) partial carotid occlusion; (C1 and C2) anterior cerebral artery occlusion; (D) partial basilar artery occlusion; (E1 and E2) middle cerebral artery occlusion. Digital compression of the contralateral carotid usually produces a syncopal state with clonic convulsive movements greatest on the side opposite the compression when an occlusive lesion

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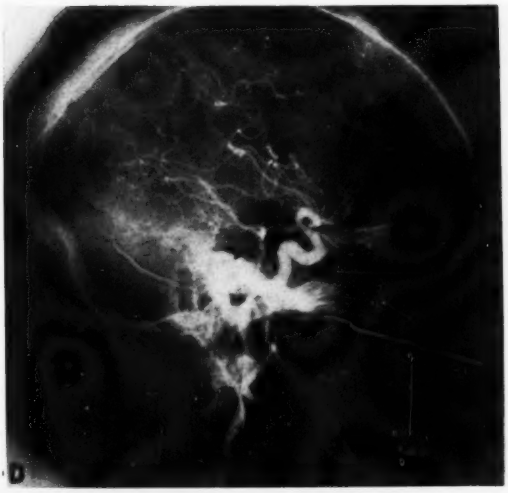

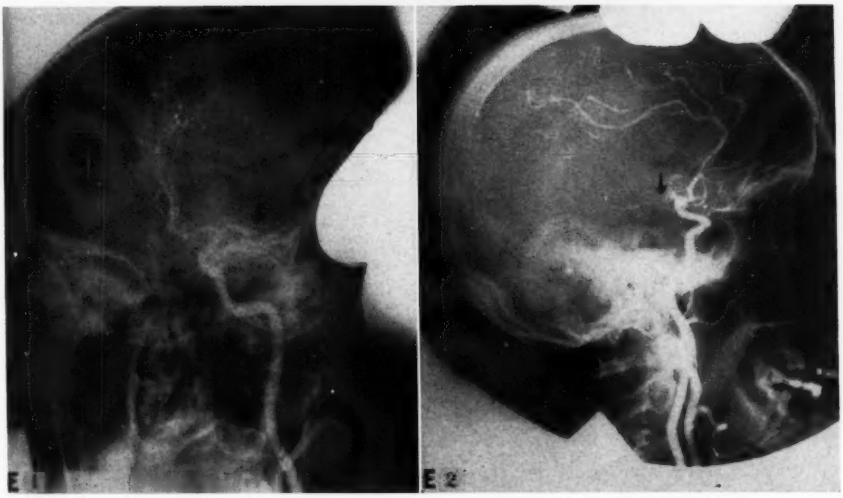

(A, B and C) is recently present. (A sensitive carotid sinus reflex may co-exist with the occlusive disease. Syncope upon compression will still occur after atropine gr. 1/100.) When basilar artery occlusion is present as in D, compression of either right or left carotid may produce syncope. Occlusion of the trunk of the middle cerebral artery as in E or the terminal branches (normal angiogram) is not associated with syncope upon compression of the contralateral carotid.

TABLE 1

Classification of results of digital carotid compression in 100 patients with hemiparesis or hemiplegia of varying origin

I. Syncopal Responses

A. Carotid occlusion (with hemiplegia—hemiparesis)—29 cases

1. Complete unilateral occlusion—17

Syncope	No syncope
13	4 (1 with bilateral occlusion but with good collaterals)

2. Incomplete unilateral occlusion (insufficiency syndrome)—12 cases

Syncope	No syncope
11	1

Comment: Two cases in which there was no response by syncope in group 1 were of long standing (7 months, 1½ years) as was the 1 case in group 2.

B. Basilar occlusion (with hemiplegia—hemiparesis)—7 cases

Syncope	No syncope
6	1

Comment: The syncopal response in this group occurred either upon right or left compression. This response was similar to that of 2 patients with bilateral incomplete carotid occlusions. The exception was a patient with partial occlusion of 2 years' duration.

C. Anterior cerebral artery occlusion (usually hemiparesis greatest in upper extremity)—10 cases

Syncope	No syncope
9	1

Comment: Patients having this lesion without paralysis also were found to develop syncope when the contralateral anterior cerebral was temporarily occluded by carotid compression.

II. Non-Syncopal Responses

D. Middle cerebral artery occlusion—10 cases

Syncope	No syncope
3	7

Comment: One of the patients in the syncopal group also had an occlusion of the anterior cerebral artery. One in the non-syncopal group had a chronic carotid occlusion with an acute middle cerebral artery occlusion.

E. Arteriosclerosis of small arteries or emboli with ischemia or infarction (normal angiogram)—10 cases

Syncope	No syncope
0	10

F. Mass lesions: brain tumor, hematoma (with hemiplegia—hemiparesis)—26 cases

Syncope	No syncope
6	20

Comment: Of the patients responding by syncope 1 presented a sensitive carotid sinus; 1 was 70 years of age; 2 had papilledema. (One patient with a chronic subdural hematoma also responded by syncope.)

G. Encephalitis—meningitis—multiple sclerosis—trauma (with hemiparesis)—8 cases

Syncope	No syncope
0	8

Note: In patients in whom syncope was expected (46) there were exceptions or misleading responses in 7, or 15 per cent. In patients in whom syncope was not expected (54) there were exceptions or misleading responses in 9, or 16 per cent. The factors influencing the response include length or time of occlusion (collateral circulation), the presence of a sensitive carotid sinus, increased intracranial pressure or a partial carotid occlusion accompanying other lesions. Evaluation of these factors increases the accuracy in evaluating the compression test.

Heymans and associates (1929) focused attention upon a neural reflex mechanism in the "carotid sinus" to regulate blood flow to the brain. Weiss and Baker then popularized the clinical aspects of the sensitive "carotid sinus" reflex mechanism. The "cerebral type" of this reflex (with absent or minimal cardiac response) was not clearly defined and the syncope induced was thought to be caused by cerebral vasoconstriction.

Presently clinical evidence turns attention back to cerebral ischemia as a cause for syncope with convulsive movements when carotid compression is employed. In patients with recent occlusive cerebrovascular disease involving the carotid artery (completely or partially) or the anterior cerebral artery, digital compression of the contralateral carotid may induce cerebral ischemia and thus result in syncope and convulsive movements. Anatomical abnormalities of the anterior or posterior communicating arteries may also play a role in this response. Neurologic symptoms also may be increased. When the vertebral-basilar system is partially occluded, compression of either carotid artery may result in syncope.

It appears that blood flow to the brain is significantly determined by occlusive atheromatous disease of the trunk vessels which may be reduced at single or multiple points. A borderline sufficient flow may be rendered acutely insufficient by compression of the carotid artery. Such compression may serve as a test for occlusive cerebrovascular disease. It has proved to be safe not only in the past but also during its present usage in hundreds of patients.

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SACCULAR ANEURYSMS OF THE POSTERIOR FOSSA*

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OUR interest in aneurysms of the posterior fossa has been stimulated recently by 2 cases in which the lesions were diagnosed and successfully treated by operation within a 6-month period. A review of the literature revealed only 2 other patients who had been successfully treated surgically.[9,11] Both of these were operated upon without the aid of pre- or postoperative vertebral angiograms. A few additional cases have been reported to us in personal communications[3,8] but the number of successfully treated aneurysms of the posterior fossa is still quite limited.

It has been estimated conservatively that at least one-sixth of intracranial aneurysms arise from the basilar-vertebral system and it is, therefore, our conviction that more of these would be diagnosed if they were searched for more diligently. Perhaps the feeling among neurosurgeons that little could be done for aneurysms of the posterior fossa even if they were diagnosed has been the factor deterring a more aggressive search for these aneurysms. It is our belief that vertebral angiography is reasonably safe and that it should be considered more frequently. Schultz[10] has recently reported approximately 50 vertebral angiograms, all of which were done without complications.

Fulfilling Dandy's[4] prediction, Schwartz,[11] in 1948, reported the first successful direct surgical cure of a small sacculated arterial aneurysm in the posterior fossa.[6,13] It arose from an abnormal artery in the left cerebellopontine angle and measured about 4 mm. in diameter. The patient had suffered several episodes of subarachnoid hemorrhage with subsequent cranial nerve signs. The aneurysm was trapped between two clips without residual signs.

Rizzoli and Hayes,[9] in 1953, reported the second successful case. Their patient was admitted with subarachnoid hemorrhage without localizing signs and failed to respond to conservative therapy. Subsequently, bilateral occipital and temporal burr holes were placed; the findings were negative. Finally a ventriculogram suggested a lesion in the posterior fossa. The patient had dilated ventricles with a shift of the fourth ventricle to the right. An exploration of the posterior fossa demonstrated an aneurysm, 2 cm. by 1 cm. in size, arising from the left posterior inferior cerebellar artery just beneath and anterior to the left cerebellar tonsil. This was trapped and excised. During a stormy postoperative course the patient had bilateral ataxia of the

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arms, weakness of both legs, and confusion, but he slowly improved. At the time of the report, 5 years after surgery, he was gainfully employed and his only residual sign was ataxia of the left arm.

In the cases reported here the diagnoses were made by percutaneous vertebral angiography and we believe they emphasize the necessity of complete cerebral angiography in cases of unexplained subarachnoid hemorrhage. The carotid angiograms were done with local anesthesia; however, for the vertebral angiograms general anesthesia was used. Angiography was carried out percutaneously using Hypaque Sodium, which we believe to be the medium of choice.[15]

CASE REPORTS

Case 1. #222492. K.G., a 48-year-old white male mechanic, was admitted to the Neurosurgical Service of the Baptist Memorial Hospital on July 18, 1956, complaining of severe headaches, stiffness of the neck, and photophobia. Eleven hours prior to admission he was awakened from sleep by severe occipital pain radiating into the parietotemporal region bilaterally. He also noted tingling dysesthesias, weakness of the left extremities and blurred vision of the left homonymous visual field. This persisted for about 15 minutes. Subsequently, there developed nausea, vomiting, stiffness of the neck, drowsiness, and mild disorientation. Previous history revealed that he had experienced intermittent headaches for about 6 months. For the past 10 years he had had bilateral Dupuytren's contractures which seemed to be progressive.

Examination. The vital signs were normal. He was slightly confused. He had signs of meningeal irritation and examination of the optic fundi revealed bilateral subhyaloid hemorrhages. A lumbar puncture yielded grossly bloody spinal fluid under a pressure of 300 mm. of water. His bleeding and clotting times were within the limits of normal.

His condition remained stable and since he did not appear to be in critical condition, bilateral carotid arteriograms were done the following day. These were normal. Two days after admission right vertebral arteriography was performed. A saccular aneurysm arising from what was considered to be the right posterior inferior cerebellar artery was demonstrated.

1st Operation. On July 23, 1956, under general anesthesia, a right suboccipital craniectomy was performed. Exploration of the posterior fossa inferiorly revealed signs of subarachnoid hemorrhage. Dissection was then carried rostrally and a small mass of clotted blood was found beneath the arachnoid in the region of the 7th and 8th cranial nerves. An attempt to free up the arachnoid by gentle dissection was made but sudden brisk bleeding obscured the field. This was controlled by local pressure and Gelfoam. A silver clip was placed on a vessel presumed to be the anterior inferior cerebellar artery, which seemed to be entering the bleeding area. It was felt inadvisable to continue further operative manipulations and the wound was closed.

Postoperatively the patient had diplopia caused by paralysis of the right 6th nerve, a transient right peripheral facial paresis and deafness in the right ear. There were deviation of the uvula to the left, vertigo and ipsilateral ataxia. He was somewhat confused for a few days but rapidly recovered and was discharged 8 days postoperatively. The only deficit on discharge was vertigo on sudden motion and deafness on the right.

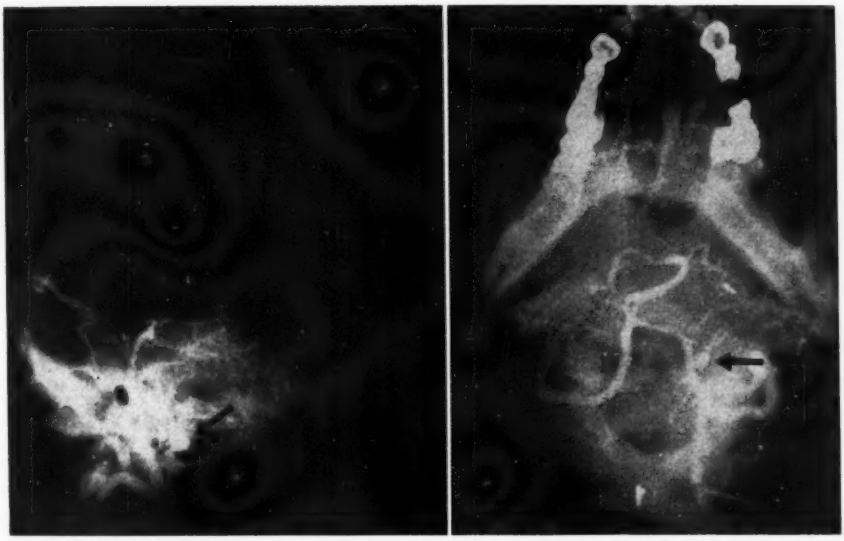

FIG. 1. *Case 1.* Right vertebral angiogram demonstrating aneurysm of posterior inferior cerebellar artery.

Readmission, Aug. 21, 1956. The patient stated that he had done quite well until 3 days previously when he suddenly felt hot, cold, became drowsy, nauseated, and had bilateral occipital pain.

Examination. His vital signs were essentially normal. There were signs of meningeal irritation and continued deafness on the right. A diagnosis of recurrent subarachnoid hemorrhage was made. On Aug. 22, 1956, right vertebral arteriography was repeated and again the aneurysm in the region of the posterior inferior cerebellar artery was demonstrated (Fig. 1).

2nd Operation. On Aug. 27, 1956, under general anesthesia, suboccipital craniectomy was again performed. There were signs of recent subarachnoid hemorrhage. The cerebellar hemisphere was elevated and the posterior inferior cerebellar artery was identified. The aneurysm arose from this tortuous vessel. It lay against the medulla beneath the rootlets of the 9th and 10th nerves, measured approximately 5 mm. in diameter, and was surrounded by adhesions. It was trapped between Olivecrona clips. Further exploration revealed no abnormality.

Postoperatively the patient had severe horizontal nystagmus, right facial paresis, deviation of the uvula to the left, brassy speech, dysphagia, vertigo, ipsilateral cerebellar ataxia and diplopia. He was again quite confused for a few days but then slowly improved. By the 6th day his condition seemed much better and right vertebral arteriography was repeated; this confirmed the absence of the lesion (Fig. 2).

At the time of discharge from the hospital on Oct. 10, 1956, his deficit consisted of mild ataxia, slightly brassy speech, 6th nerve weakness on the right, and nerve deafness on the right. When he was last examined in March 1957, he had mild diplopia on extreme right lateral gaze, deafness on the right, elevation of the uvula to the left, and very mild ataxia. He walked without difficulty and had had no further symptoms.

FIG. 2. *Case 1.* Right vertebral angiogram demonstrating successful clipping of aneurysm.

Case 2. #25061. C.N., a 33-year-old white farmer, was admitted to the medical service of the City of Memphis Hospitals on Jan. 17, 1957, in a state of agitated delirium. Relatives stated that 72 hours previously he awakened from sleep complaining of severe headaches, became nauseated, vomited and had stiffness of his neck. Twelve hours prior to admission he became very confused and disoriented.

Past history revealed that at the age of 6 he had suffered an injury to the left eye with a stick and subsequently the pupil became enlarged, with ptosis of the lid and impairment of movement. He had received a cerebral concussion and fracture of the right tibia in an automobile accident in 1941.

Examination. Temperature was 100°F., pulse rate 68, respiratory rate 24, and blood pressure 180/80. He was well developed, slightly dehydrated, and extremely restless. Neither pupil reacted to light. The left pupil was fixed and dilated and held in a neutral position. Corneal sensation was normal. His neck was slightly hyperextended. Deep tendon reflexes were hyperactive. The remainder of the findings were normal except for bilateral ankle clonus. A lumbar puncture revealed grossly bloody spinal fluid under pressure in excess of 600 mm. of water. The supernatant fluid was found to be xanthochromic after centrifugation. Roentgenograms of the skull and chest were normal. Blood and urine were normal. Neurosurgical consultation was obtained 12 hours after admission. Bilateral carotid arteriograms were done and these were normal.

Course. A conservative regimen was instituted and the spinal fluid gradually cleared. On the morning of Jan. 28, 1957, he again became irrational and disoriented, and complained of pain in his head and neck. It was felt that he had suffered a recurrent subarachnoid hemorrhage. That afternoon right vertebral arteriography was performed and a saccular aneurysm arising from the right posterior inferior cerebellar artery was demonstrated (Fig. 3).

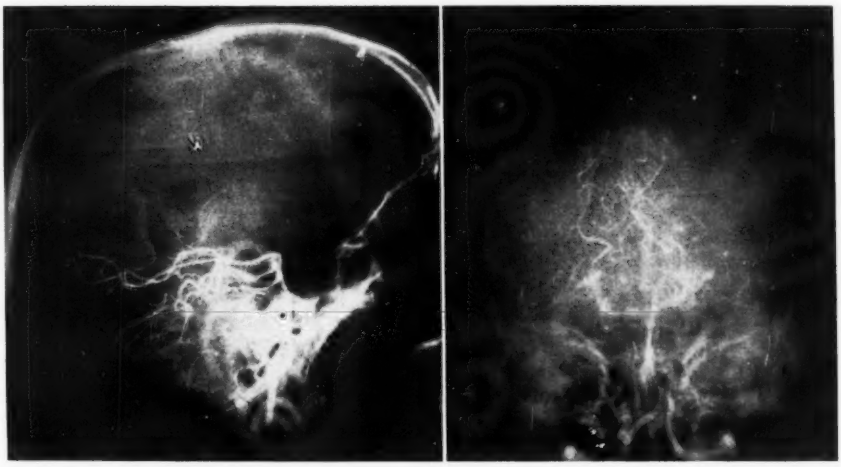

FIG. 3. *Case 2.* Right vertebral angiogram demonstrating aneurysm of posterior inferior cerebellar artery.

Operation. On Jan. 30, 1957, under general anesthesia, a right suboccipital craniectomy was performed. Exploration of the posterior fossa revealed evidence of recent subarachnoid hemorrhage. The cisterna magna was entered; the cerebellar hemisphere was retracted and a saccular aneurysm, measuring about 0.5 cm. by 1.5

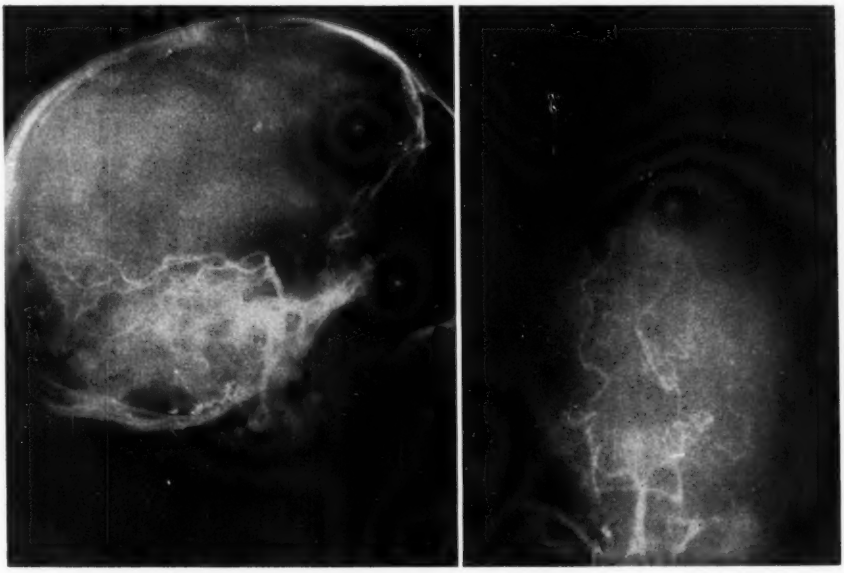

FIG. 4. *Case 2.* Right vertebral angiogram demonstrating successful clipping of aneurysm.

cm., was exposed. It was being demonstrated to the staff on the medical service when it suddenly ruptured. The hemorrhage was controlled by direct finger tamponade and Arfonad-induced hypotension. The usual silver clips and Olivecrona clips were placed proximal and distal to trap the aneurysm. As a result of the bleeding which obscured the field several other vessels were inadvertently torn and bleeding from these vessels was controlled with silver clips.

The *postoperative course* was quite stormy, with fluctuations of sensorium and vital signs. He became quite confused and had evidence of an agitated psychosis. The right extremities were moderately ataxic. On Feb. 3, 1957, a lumbar puncture revealed bloody spinal fluid with xanthochromia. The pressure was 125 mm. of water. Pneumonitis of the right lower lobe complicated the immediate postoperative course but this finally cleared. By the 14th postoperative day the spinal fluid was clear and the patient's sensorium was normal. The ataxia progressively improved.

On Feb. 22, 1957, right vertebral arteriography was repeated and this demonstrated that the aneurysm had been successfully trapped (Fig. 4). The patient was discharged the following day with only a residual of mild ipsilateral ataxia.

When he was last examined on March 21, 1957, he was walking with crutches because of an incompletely healed fracture of his right tibia. He had no complaints but there was a very mild ataxia of the right lower extremity. There was no ataxia of his arms. However, this patient was quite nervous at the time of our examination, as he had been all his life, and it is possible that a very mild ataxia could have been masked by his nervousness. There was no change in his cranial nerve findings.

DISCUSSION

A review of the literature reveals only a few successful surgical attacks upon saccular arterial aneurysms of the posterior fossa.[9,11] However, it has indicated the feasibility of this procedure. Successful attack of aneurysms of the posterior fossa is limited by accessibility. Uihlein and Hughes[14] reported the surgical exploration of 14 basilar-vertebral aneurysms on which no definitive therapy could be rendered. Subsequently 8 of the 14 patients succumbed to ruptures. Treatment of aneurysms of the large vessels of the posterior fossa, such as the vertebral artery, will probably remain conservative as exemplified by Steelman,[12] who relieved complaints of intolerable bruit and occipital headaches by re-enforcement of a vertebral aneurysm with muscle.

We were able to learn of several more aneurysms which had not been reported in the literature.[3,8] Poppen has successfully attacked three aneurysms of the posterior inferior cerebellar and one aneurysm of the anterior inferior cerebellar artery. Bassett,[2,3] discussed the embryological origin of aneurysms and mentioned that he had treated an aneurysm of the mid-portion of the left posterior inferior cerebellar artery successfully.

It is our feeling that vertebral arteriograms should be resorted to more frequently when subarachnoid bleeding cannot be accounted for by aneurysms of the carotid system. Surgical management of these lesions should be direct excision, trapping, or clipping when feasible. The role of various reenforcement procedures remains to be seen. Ligation of the vertebral artery does not seem indicated. Although vertebral ligation has been accomplished, the experiences of Dandy[4] and French and Haines[5] furnish adequate

evidence of the danger involved. In addition Bakay and Sweet[1] have shown that ligation of one vertebral artery does not reduce distal intravascular pressure providing both vertebral arteries are patent.

The risk involved with sacrifice of vessels of the posterior fossa must be weighed against the risk of recurrent subarachnoid hemorrhage. Occlusion of the posterior inferior cerebellar artery does not seem to have any serious sequellae. Lewis *et al.*[7] in a follow-up of 20 clinically diagnosed cases of thrombosis of the posterior inferior cerebellar artery reported good results from 6 months to 12 years after the accident. There seemed to be only 2 cases of deaths which were caused directly by occlusion of the posterior inferior cerebellar artery. The experience with our 2 cases would lead us to believe the residual defects from clipping the posterior inferior cerebellar artery are quite minimal and it is our feeling that they are preferable to the risk of repeated subarachnoid hemorrhage.

In conclusion it is our feeling that vertebral arteriography should be resorted to more frequently and that a more aggressive approach should be made toward aneurysms in the posterior fossa.

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CAVERNOUS HEMANGIOMAS OF THE CEREBRAL HEMISPHERES*

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THE classification of vascular lesions of the brain is a very controversial topic. In the literature the same type of tumor may be listed by various authors under different names. In this paper 3 cases of well encapsulated vascular tumors, "cavernous hemangiomas," are presented. The differentiation of these tumors from other vascular lesions is most important clinically because of the ease of successful surgical removal.

CASE REPORTS

Case 1. B.T., 33 years old, a right-handed woman, was admitted to the University of Michigan Hospital on Sept. 11, 1956 because of headaches. Seven years previously, at the time of the birth of her first child, she began to have throbbing headache over the vertex which radiated forward to the mid-frontal area. Intermittently the headache was accompanied by nonprojectile vomiting, true vertigo, and transient loss of vision. Her physician made a diagnosis of migraine headaches and treated her with ergotamine with some symptomatic relief. Severe headaches occurred four to five times a year and were accompanied by a residual pain in the left temporal area. Fourteen months before admission she began having generalized tonic and clonic seizures lasting about a minute. These seizures were preceded by a premonitory headache and were followed by transient visual loss and a postictal stupor of half an hour's duration. There was also mental confusion. During the 2 months prior to admission she had had impairment of memory and difficulty in expressing herself. Her menses had been irregular and she had been told that she had a uterine fibroid.

The only positive findings on neurological examination were a Grade 1 papilledema bilaterally and a partial left homonymous hemianopsia. On physical examination a mass was noted on the anteromedial portion of the left tibia which the patient stated had been present for a period of 13 years. The lump on the tibia had become painful at the time of her menstrual periods. Roentgenograms of the left tibia revealed a thickening of the periosteum immediately overlying this mass. An electroencephalogram revealed a right posterior temporoparieto-occipital focus which was suggestive of a malignant neoplasm. A right internal carotid arteriogram showed evidence of a right posterior temporoparietal brain tumor. Unfortunately, the lateral view of the arteriogram has been misplaced and cannot be reproduced in this paper. Roentgenograms of the chest were negative.

On Sept. 17, 1956, ventriculography was attempted and upon inserting a ventric-

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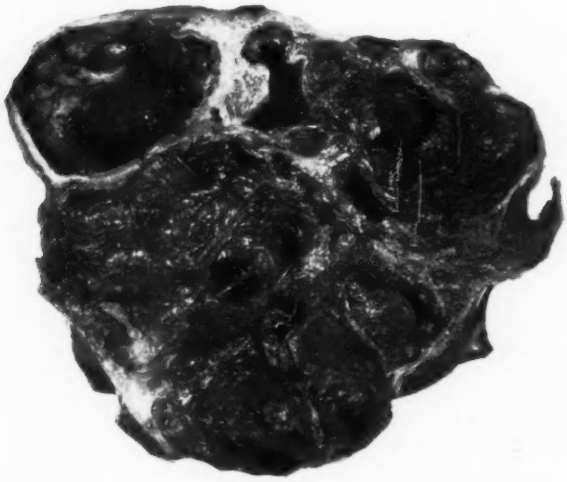

FIG. 1. *Case 1.* Gross appearance of the tumor.

ular needle at the right posterior parietal area to tap the ventricle, a firm mass was encountered. A right temporoparietal osteoplastic craniotomy was performed with excision of a well encapsulated mass, 6 cm. by 8 cm. in size (Fig. 1). The lesion was noninvasive and had very few blood vessels entering the capsule. It was easily "shelled out."

On Oct. 3, 1956 the lump over the left tibia was biopsied and the pathologist reported a fibrohemangioma.

The patient was discharged Oct. 7, 1956. She had a complete left homonymous hemianopsia as her sole residual neurological deficit.

Gross and Microscopic Description. The specimen was covered by a thick fibrous smooth membrane with several dome-shaped exuberances. The cross section showed numerous cavities of various size which were separated by thick septa. Microscopic

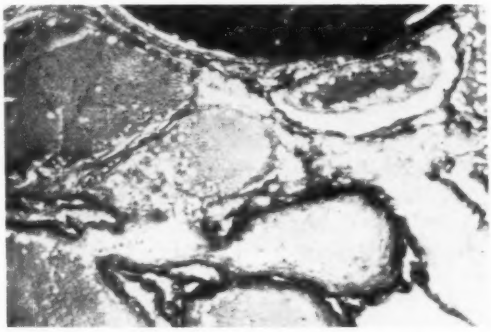

FIG. 2. *Case 1.* Small blood-filled cavities lined with endothelium.

examination revealed small and large cavities which were thin-walled, lined by endothelium and filled with blood (Fig. 2). The septa and capsule showed evidence of histiocytes which were filled with hemosiderin, and also areas infiltrated by lymphocytes.

Case 2. R.H., 33 years old, a right-handed woman, was admitted to the University of Michigan Hospital on Oct. 6, 1956 with a chief complaint of suboccipital headache and convulsive seizures. She originally noted the onset of her headache following her first pregnancy 7 years previously. Two months before entering the hospital she had an episode of vomiting, following which she became unconscious. A similar attack which lasted 30 minutes occurred in the hospital. She assumed an opisthotonic position.

Her blood pressure was 140/60, the pulse rate was 40 to 60 per minute, and the respirations were 20 per minute. She was confused and readily distracted although moderately coherent in most of her answers to questions. On neurological examination there were the following abnormal findings: bilateral papilledema, left pupil larger than the right, left central facial weakness, and bilateral pyramidal tract signs in both upper and lower extremities.

Routine roentgenograms of the skull demonstrated erosion of the posterior clinoid processes. Ventriculography was performed within 2 hours after admission to the hospital; the right ventricle was encountered at a distance of 2.5 cm. below the inner table of the skull. The ventricular pressure was 450 mm. of water. The left ventricle, however, was tapped successfully at 4.5 cm. from the surface. Indigo carmine which was injected into the right lateral ventricle was recovered on tapping the left one, indicating that there was no block between the lateral and 3rd ventricles. The right parietal bone was extremely thin. Ventriculograms revealed evidence of a large tumor which seemed to be within the right lateral ventricle.

A right temporoparietal osteoplastic craniotomy was performed and a well encapsulated tumor, 10 cm. in diameter, was totally removed from the right lateral ventricle. There was almost a complete absence of blood vessels entering the tumor so that the mass could be readily packed off and removed in toto through a relatively small cortical incision.

The postoperative course was complicated by an unexplained fever for about 10 days after operation, but this finally subsided. At the time of the patient's discharge on Oct. 21, 1956 she was mentally alert and the only residual neurological finding was a complete left homonymous hemianopsia.

Gross and Microscopic Description. The encapsulated tumor measuring, after fixation, $8 \times 6 \times 5\frac{1}{2}$ cm., had a smooth shiny surface (Fig. 3). Cross section showed numerous cavities. There was microscopic evidence of hemorrhage into the capsule and in the septum. The septum was lined with endothelium and fibrocytes. There was a large amount of hemosiderin in the tissue.

*Case 3.** "L.O'T., a white female child, aged $2\frac{1}{2}$ years, entered the University Hospital April 30, 1937, because of convulsions. History revealed that at the age of twenty months the patient developed weakness of the left leg which gave rise to the diagnosis of a left spastic monoplegia. Six weeks prior to admission, there was noted increasing weakness of the left leg, abnormal salivation, persistent vomiting of a

* Woods and Kahn[8] reported this case in 1942. The authors wish to express their appreciation to Drs. Woods and Kahn and the publishers of *Surgery* for the opportunity of reprinting the case.

FIG. 3. *Case 2.* Gross appearance of the tumor.

nonprojectile type, and frequent convulsions, especially while asleep, characterized by grinding of the teeth, "stiffening out," and rolling of the eyeballs. During an attack witnessed in the hospital, there was twitching of the face and extremities for forty-five seconds, with unconsciousness. Significant findings were abnormal drooling of saliva, spastic weakness of the left leg, and extensor plantar response on the left. Funduscopic examination was negative. Pneumo-encephalogram revealed evidence of a space-occupying lesion deep in the right temporal lobe. At operation an encapsulated tumor, approximately the size of a golf ball, was encountered by a transcortical incision through the mid-temporal region 2 cm. in depth. The entire tumor was removed [E.A.K.]. The ventricle was not entered. The dura was completely closed and the bone flap replaced. Pathologic diagnosis was cavernous hemangioma.

"Postoperatively the patient showed a marked weakness of the left arm and leg which gradually improved. She was discharged on the sixteenth postoperative day with a mild left hemiparesis."

Subsequently the patient had left-sided Jacksonian seizures and was readmitted to University Hospital for lysis of adhesions by subdural air injection. This procedure was employed by Woods and Kahn in the hope of preventing the reformation of corticomeningeal adhesions and thus avoiding convulsive episodes. The patient was free of convulsive seizures for a period of $1\frac{1}{2}$ years, and then had a recurrence of them although they were of less severity.

Microscopic Description. The tumor was composed of numerous small vascular spaces of varying sizes which were lined by endothelium but no evidence of normal vascular structures was found (Fig. 4). Some of the cavities near the surface were thrombosed (Fig. 5). The wall of the angiomatous cavity was formed by endothelium with numerous fibers of reticulin (Fig. 6).

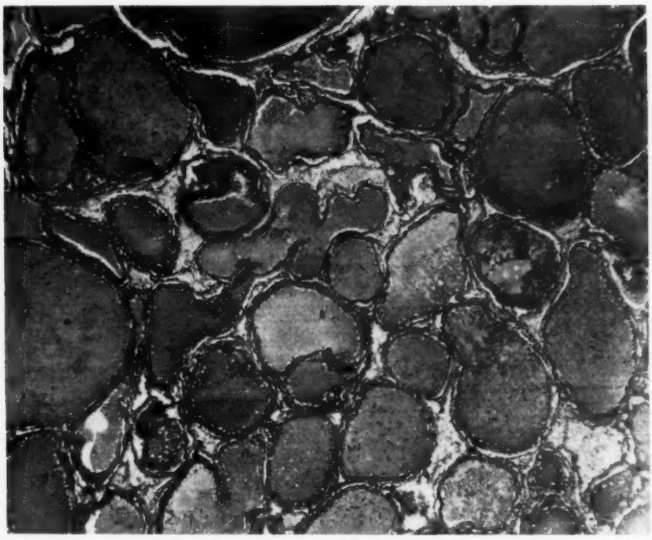

FIG. 4. *Case 3.* Small blood-filled vascular spaces.

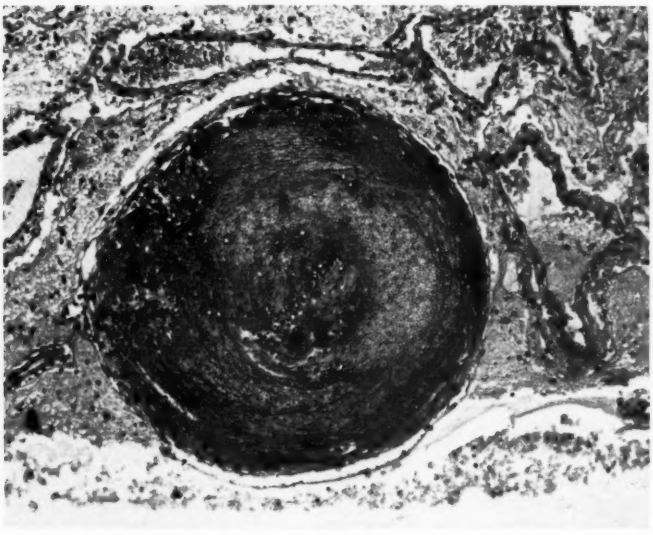

FIG. 5. *Case 3.* Cavity filled with thrombosed blood.

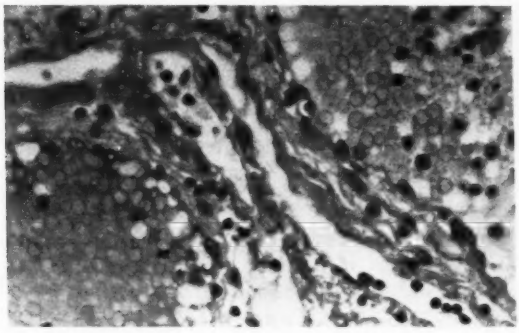

FIG. 6. *Case 3.* Vascular cavities lined with thickened endothelium.

COMMENT

These cases are rather rare. In the entire brain tumor series at the University of Michigan Hospital there have been but 3 intracranial tumors of the cavernous hemangioma type recorded. In Cases 1 and 2 the patients were of the same age, their lesions were in identical locations, both had the onset of symptoms in association with a pregnancy 7 years before admission to the hospital, and both had removal of the lesion in toto with no residual neurological findings except a complete left homonymous hemianopsia.

The patient in Case 1 had the additional finding of a hemangioma involving the left tibia. Perhaps if this had been biopsied prior to craniotomy one might have considered intracranial cavernous hemangioma in the differential diagnosis.

Case 3 represents a similar lesion of smaller size in a younger age group. Its location was practically identical with that of the lesions in Cases 1 and 2.

Pathology. Cases 1 and 2 have almost identical histological appearance. They can be diagnosed as cavernous hemangiomas although the probability that they represent an arteriovenous aneurysm cannot be positively eliminated. The cavities filled with blood are lined by thin endothelium.

Case 3 presents the same diagnostic problem, but it differs from the first 2 cases since this tumor is formed by multiple small vascular spaces not separated from each other by septa. It is possible that all 3 cases do not represent tumors in a true sense but malformations (arteriovenous aneurysms).

DISCUSSION

Cavernous hemangiomas of the cerebellum are occasionally associated with Lindau's disease. It is difficult to determine how frequently the cavernous hemangiomas do occur in the hemispheres since there is little consensus among the authors as far as the terminology of the vascular tumors in the brain is concerned. In 1945, Noran[5] made an attempt to reclassify intra-

cranial tumors and malformations. The value of his contribution is increased by the fact that he discussed all existing classifications.

In the Cushing and Bailey[3] series 2 tumors are listed which are grossly and histologically similar to those described in the present study; they are Cases 15 and 17 both found in the cerebellum and have, therefore, no correlation with our cases.

Dorothy Russell[6] stated that 7 cavernous hemangiomas are on file at the Institute of Pathology in London. She described them as a nonencapsulated system of cavernous and capillary spaces. In her opinion it is not certain whether they represent true tumors or vascular malformations.

This opinion is shared by Louise Eisenhardt.[4] In a personal communication she states, "The histology of cavernous angioma in cases reported in the literature is inadequate in most instances to determine whether some of these lesions are actually telangiectases or vascular malformations."

Bergstrand, Olivecrona and Tönnis[2] listed several cases of cavernous hemangiomas found in the literature. Those from their own series are described as well limited tumors without a capsule which are composed of cavities of varying size.

Astwazaturoff's[1] case reported in 1910 has the greatest similarity to those described in our present study. Histologically the tumor described by him has a thin capsule which is of varying thickness and forms a meshwork, in between which there is mostly coagulated blood.

Wolf and Brock,[7] in describing the pathology of cerebral angiomas in 1935, did not report any well encapsulated lesions.

In the present study only in Case 1 was there another cavernous hemangioma and this was on the tibia; in the other 2 cases there were only solitary brain tumors. Specimens from Cases 1 and 2 have a thick capsule and are formed by large blood-filled spaces which are separated by thick connective-tissue walls. Microscopically, a large amount of small blood-filled spaces also can be found which mostly are located adjacent to the capsule or septa. In the thick walls of the capsules and the septa there was evidence of old and recent hemorrhage, probably per diapedesis, and elements of chronic irritation were found. A small number of large cavities were filled with thrombosed blood.

Case 3 represents a cavernous hemangioma, well encapsulated and without a thick septum. The cavities are very small and separated from each other by a thickened endothelial membrane.

SUMMARY

Three cases of well encapsulated lesions, cavernous hemangiomas, of the cerebral hemispheres are presented in detail giving their clinical and pathological features. The importance of accurate diagnosis in these cases is stressed because of the possibility of successful surgical treatment.

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ARTERIAL ANEURYSMS OF THE INTERNAL CAROTID ARTERY AND ITS BIFURCATION

AN ANALYSIS OF 69 ANEURYSMS TREATED MAINLY BY DIRECT SURGICAL ATTACK

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RECENT experience with surgical treatment of patients with ruptured intracranial arterial aneurysms who have survived the acute stage of bleeding shows that although the mortality is sometimes comparatively high, it is still considerably lower than the mortality for conservatively treated patients.[8,10,12] Instead of the question whether to operate or not, new problems arise; some of them are common to all aneurysms, others are caused by the different locations of the lesion. Consequently, there are reasons for analyzing the aneurysms in groups instead of treating them as an entity. The initiative was taken in 1953 by Norlén and Barnum[11] with an analysis of aneurysms arising from the anterior communicating artery, and Black and German[2] with a study on aneurysms of the internal carotid artery. Analyses of separate groups of aneurysms have also been made by several other authors.[5–8,14,15] Recently Harris and Udvarhelyi[9] have published a series of 90 aneurysms arising at the internal carotid-posterior communicating artery junction. The majority of their patients were treated with carotid ligation.

This report concerns patients with saccular nonfistulous aneurysms arising from the internal carotid artery or its bifurcation and diagnosed in the Finnish Red Cross Hospital.

MATERIAL

Up to December 31, 1956 a total of 195 arterial saccular aneurysms were found in 184 patients. Of these, 7 patients had 2 aneurysms and 2 patients had 3 aneurysms; 1 of the latter had all 3 aneurysms on the same internal carotid artery. One patient had a saccular aneurysm and an arteriovenous malformation.

There were 132 patients submitted to surgery, more than 95 per cent of them by the intracranial approach. The distribution of the aneurysms and the number operated upon in the different groups is shown in Table 1.

The total number of aneurysms on the internal carotid artery or its bifurcation was 69 in 65 patients, 2 of whom had bilateral aneurysms and 1 had 3 aneurysms on the same side. This group covers roughly 35.5 per cent of the total number of aneurysms verified in the Finnish Red Cross Hospital. The percentage of aneurysms of the middle cerebral artery was 28 (55

TABLE 1

Location of aneurysms in material of Finnish Red Cross Hospital 1937–1956

Location	Total Number of Aneurysms	Number of Aneurysms Operated Upon
Internal carotid artery	60	43
Carotid bifurcation	9	7
Anterior cerebral artery	16	11
Anterior communicating artery	41	29
Pericallosal artery	10	7
Middle cerebral artery	55	35
Vertebral system	4	—
Total	195	132

aneurysms); the group with aneurysms of the anterior cerebral and anterior communicating arteries was almost the same size (57 aneurysms, 29 per cent).

SEX AND AGE

In the total material there were 35 females and 30 males. The age distribution is shown in Table 2 (age on admission being approximately the same as the age at onset of symptoms).

The age of the patients ranged from 11 to 68 years, the average being 41.5 years.

INCIDENCE OF SUBARACHNOID HEMORRHAGE

One patient had 4 attacks of bleeding before operation, 2 had 3 attacks, 17 had 2 and 41 had 1 episode of bleeding. Four patients apparently had not bled. These 4 and 10 other patients (14 patients) had ophthalmoplegia on admission to the hospital. Four patients were totally hemiplegic, 2 of them also aphasic, and another 4 patients were hemiparetic.

LOCATION OF ANEURYSMS

The closer location of the 69 aneurysms is shown in Table 3.

Of the supraclinoid aneurysms 1 pointed frontally, 3 laterally and the rest posteriorly.

The number of infraclinoid aneurysms is rather small compared with

TABLE 2

Sex and age distribution of patients with aneurysms of the internal carotid artery

	10–19	20–29	30–39	40–49	50–59	60–	Total
Males	1	4	9	10	5	1	30
Females	1	2	9	13	10	—	35
Total	2	6	18	23	15	1	65

TABLE 3

Closer location of internal carotid artery aneurysms

Location	Right	Left	Total
Internal carotid artery, infraclinoid	2	3	5
Internal carotid artery, supraclinoid	29	26	55
Carotid bifurcation	3	6	9
Total	34	35	69

many other series, the ratio being 5 infraclinoid to 55 supraclinoid aneurysms. Steelman *et al.*[16] reported a series with a ratio of 21 infraclinoid to 20 supraclinoid aneurysms, and Graf,[6] 5 to 25.

ANEURYSMS NOT OPERATED UPON

On 17 patients no surgery was done. Some of these patients had refused to submit to operation and the hospitalization of the rest occurred before the commencement of active aneurysmal surgery. The fate of 14 of these patients treated before the spring of 1955 has been reported in a separate paper.[1] Of these, 11 died during the follow-up period, all but 1 from recurrent hemorrhage. One of the 3 additional patients who were treated during the summer of 1955 or later, could not be traced, since he had left the country. The remaining 2 patients were living at the end of 1956, one of them with epileptic seizures (survival time after the hemorrhage $1\frac{1}{2}$ years); the other had had several recurrent attacks of bleeding but was alive at follow up 3 months after discharge from hospital.

ANEURYSMS TREATED WITH CAROTID LIGATURE

Only 4 patients were treated by ligation of the internal carotid artery. Three of them had an infraclinoid, and 1 a supraclinoid aneurysm. Two patients sustained the operation well; in the other 2 patients the ligature had to be re-opened because of hemiplegia. In spite of that, 1 of these patients died. The second patient was discharged as hemiplegic. At the follow up after $2\frac{1}{2}$ years this patient was still hemiparetic. The remaining 2 patients were doing well 6 and 5 years respectively after ligation.

ANEURYSMS TREATED INTRACRANIALLY

Since 1952 the direct intracranial attack has been the method of choice in the Finnish Red Cross Hospital. At operation the neck of the aneurysms has either been clipped, or tied with a linen thread. Only in cases in which this was not possible, was the aneurysm wrapped with hammered muscle, Gelfoam, etc. In 44 patients 46 aneurysms were attacked intracranially. The methods of treatment are shown in Table 4.

TIMING OF OPERATION

It is a well-known fact that an operation at an early stage is considerably

TABLE 4

Aneurysms attacked intracranially

Location	Method of Treatment				Total
	Thread	Silver Clip	Thread and Clip	Wrapping	
Infraclinoid	—	—	—	1	1
Supraclinoid	12	19*	4	3	38
Carotid bifurcation	3	1	—	3	7
Total	15	20	4	7	46

* 3 aneurysms clipped in 1 patient.

more dangerous than if performed later after the acute signs and symptoms of bleeding have subsided. On the other hand a deferred operation increases the risk of recurrent hemorrhage during the period of waiting. We have found the timing of the operation a minor problem because only a comparatively small number of patients have been admitted to our hospital during the acute stage of bleeding, the majority of the patients having been transferred from other hospitals 2 weeks or more after their last attack (Table 5).

The patients in the latter group were operated upon as soon as angiography had been performed; the patients in the former group, who were hospitalized during an acute episode of bleeding, were treated conservatively for 1 to 3 weeks. One of them had 3 recurrent bleedings during this period, a fact that necessitated operation a few days after the fourth hemorrhage. None of the patients in this series died in hospital while awaiting surgery. Four of the 44 patients died postoperatively in hospital, the mortality rate being 9 per cent.

CONSIDERATIONS OF TECHNIQUE

Nearly all operations have been performed using intratracheal anesthesia and controlled hypotension with Arfonad.

After frontotemporal craniotomy and removal of the arachnoid from the chiasm the internal carotid artery has been exposed up to its bifurcation. Most of the aneurysms of the upper part of the internal carotid artery pointing posteriorly or slightly laterally, a clip can often be applied across the stalk. The disadvantages of this comparatively quick and easy procedure

TABLE 5

Interval between last bleeding and operation

	Less than 1 week	1–2 wks.	2–4 wks.	1–2 mos.	2–3 mos.	More than 3 mos.	Total
Number of patients	1	2	9	17	4	11	44
Deaths	—	—	2	—	—	2	4

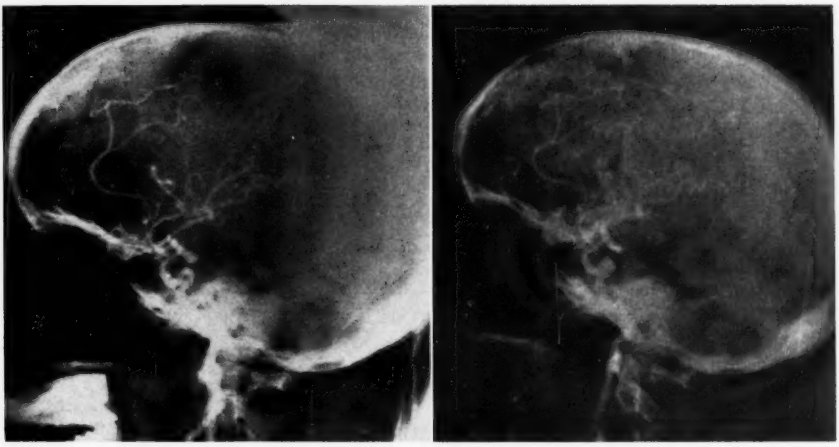

FIG. 1. Aneurysm before and after clipping. Note part of stalk still visible.

are: the risk that the aneurysm may tear when the clip is applied; the difficulty in placing the clip at right angles across the stalk, especially when the aneurysm points straight posteriorly (in a few cases a small part of the stalk was visible in the control angiogram, Fig. 1); and the difficulty in occluding a wide aneurysmal neck. Besides, there is always the risk of a secondary slipping of an apparently successfully placed clip.

Recently I have ligated the aneurysmal stalk with a linen thread whenever possible. This method is preferable insofar as the thread can always be tied close to the parent vessel and there is no danger of slipping. Applying the thread around the aneurysmal stalk has proved to be the most difficult part of the procedure, as it can seldom be done directly; generally it has been thought necessary to take the thread behind the carotid artery twice in order to catch the aneurysmal stalk in the loop. With this technique even aneurysms scarcely visible behind the carotid artery can be ligated. If the stalk lies fairly close to the carotid bifurcation the thread sometimes has to be conveyed first behind the carotid artery and then, on its way back, behind the anterior and middle cerebral arteries before the stalk can be caught. In order to reduce the risk of rupture attempts have been made not to mobilize the aneurysmal sac itself. None of the aneurysms tied with thread has ruptured during the procedure (Fig. 2).

Sometimes even the knot offers difficulties; generally it has to be tied by means of a long instrument which fixes one end of the thread under the aneurysmal stalk while the other end is being pulled cautiously.[11] In some cases when the patency of the ligature leaves some doubt, a silver clip has been applied as an extra measure to ensure safety. A control puncture of the aneurysm with a thin needle may always be advisable. In 1 case the ligature had to be left fairly loose as it threatened to occlude the internal carotid

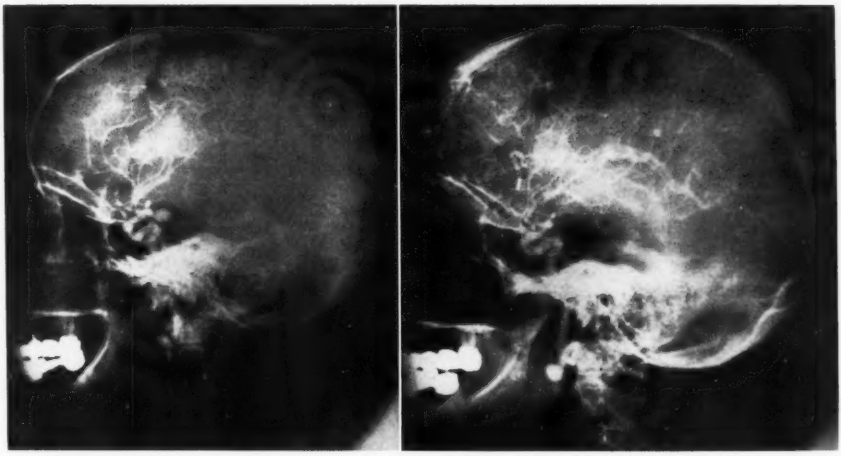

FIG. 2. Aneurysm before and after ligature with thread.

artery; in the control angiograms there was still a narrow communication between the parent vessel and the considerably diminished aneurysmal sac.

In 7 cases a thread or a silver clip could not be applied. In 1 the reason was that the aneurysm proved to lie infraclinoidally; in another that the aneurysmal wall was calcified, hard and brittle. In 2 cases of aneurysms of the carotid bifurcation, the aneurysmal stalk was so wide and adhered so closely to the middle cerebral artery, that any attempt at ligating the stalk was bound to occlude the vessel. In the 3 remaining cases the aneurysm ruptured and no clip or thread could be satisfactorily applied. In all these 7 cases the aneurysm was wrapped with muscle as effectively as possible.

A thin needle of Deschamps type was used for the thread; if it is made of soft, flexible metal the danger of lesions of the aneurysm is reduced (Fig. 3).

RESULTS OF OPERATION

The results of operation and the finding at control angiography are shown in Tables 6 and 7.

Table 6 shows that 4 patients died in hospital. In 2 of those cases a rupture of the aneurysm occurred during the operation, which necessitated a muscle packing in the first case, and in the second the bleeding could finally be controlled with clips. Both patients died within 3 days. The third patient died on the 8th postoperative day from an undiagnosed postoperative epidural clot; the fourth, a diabetic woman with a clipped aneurysm, died from a lung embolus a fortnight after the operation.

Postoperatively 2 out of the 40 survivors were hemiplegic as a result of the operation. One of these patients became hemiplegic on the evening of the day of operation, having moved her limbs on awakening from anesthesia. Control angiography revealed total thrombosis of the internal carotid

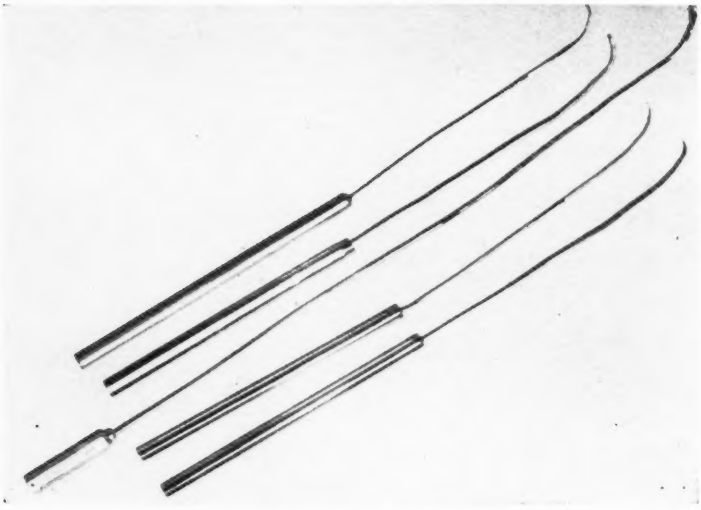

FIG. 3. Instruments used for the thread ($\frac{1}{2}$ natural size).

TABLE 6

Immediate results

Method of Treatment	Condition as Before Operation	Hemiplegia	Hemiparesis	Deaths	Total
Thread ligature	13	1	1	—	15
Silver clip	11	1*	3	3	18
Thread and clip	3	—	1	—	4
Muscle wrapping	5	—	1	1	7
Total	32	2	6	4	44

* Patient with 3 aneurysms.

TABLE 7

Findings at control angiography

Method of Treatment	Carotid Thrombosis	Aneurysm Disappeared	Part of Stalk Visible	Aneurysm Still Visible	No Angiography	Total
Thread ligature	1	13	—	1	—	15
Silver clip	—	12	2	1	—	15
Thread and clip	—	4	—	—	—	4
Muscle wrapping	—	—	—	—	6	6
Total	1	29	2	2	6	40

TABLE 8

Condition of surviving patients at follow up

Complete recovery, full working capacity	21
Impaired working capacity	
a) caused by preoperative pareses, aphasia, or ophthalmoplegia	10
b) caused by operative complications (pareses, epileptic seizures, etc.)	3
c) caused by other diseases	3
Total disability	
a) caused by preoperative hemiplegia and aphasia	1
b) caused by operative complications	—
Total	38

artery. This patient survived for 2 years with hemiplegia and epileptic fits, and died during a status epilepticus. The second postoperatively hemiplegic patient was a man with 3 aneurysms of the same carotid artery, all of which had been clipped. Control angiography showed a comparatively narrow carotid artery. He made a good recovery later.

The 6 postoperative hemipareses subsided fairly soon, mostly before the patients were discharged from hospital. One of these patients had an infection of the wound, causing necrosis of the bone flap, which had to be removed. Six months after the operation the patient was again admitted to the hospital with a brain abscess, from which he died.

FOLLOW UP

The follow-up time varies between 3 months and $4\frac{1}{2}$ years. Of the 40 patients surviving the intracranial operation, 2 died during the period of observation, as has already been mentioned. Both these deaths (1 status epilepticus, 1 brain abscess) have to be regarded as caused by the operation. The present condition of the 38 surviving patients is shown in Table 8.

On follow-up examination 6 months after operation the state of the totally disabled, preoperatively hemiplegic and aphasic patient had improved so considerably, that she has now good chance of regaining her working capacity.

Three of the patients have had epileptic seizures, 1 of them, however, only once. The comparatively low incidence of epilepsy is probably ascribable to the fact that the operation can in most cases be performed without much damage to the cerebral cortex. One patient, with a long history of epilepsy before the subarachnoid hemorrhage, had had no attacks between the operation and the follow up, 6 months later.

The results in the cases in which there was preoperative ophthalmoplegia will be discussed in a separate paper. None of these patients made a complete recovery.

COMMENTS

This study, like many others,[1,8,10,13] shows that conservatively treated

aneurysms carry a bad prognosis, with a mortality which clearly surpasses the mortality for any form of surgical treatment.

In this series carotid ligature has been used only exceptionally, and the number of cases, 4 patients, is too small to permit any conclusions. Although carotid ligature is the method of choice in cases of infraclinoid aneurysms of the internal carotid artery, there is little doubt that in aneurysms on the circle of Willis peripheral to the carotid bifurcation it is of little value, as is shown by several reports of recurrent bleedings, even fatal ones.[2,8,12,15] An intermediate position is held by the supraclinoid aneurysms of the internal carotid artery. Many authors[9,10,15] have treated aneurysms of this kind by carotid ligature with a comparatively low mortality and a very small incidence of recurrent hemorrhage. However, this study, and also others,[12] show that they may be fairly successfully treated by direct surgical attack. Even if the direct attack does not carry a lower mortality than the carotid ligature it should be preferred, as it leads to safer results and leaves the cerebral circulation of the patient unimpaired.

In this series the mortality rate for patients operated upon intracranially is 9 per cent, and the over-all mortality at follow up is roughly 14 per cent. Although this mortality cannot be considered very high, it ought to be possible to reduce it. Three out of the 4 operative deaths occurred during our first 2 years of aneurysmal surgery. After 1953 only 1 patient died, the above-mentioned diabetic with a lung embolus. However, it should be repeated that most of the patients in this series were operated on fairly late; according to McKissock's[10] classification only 1 patient belonged to his group A, 28 to group B, and 15 to group C. The number of deaths in these groups were 0, 2 and 2 respectively.

In this series hypothermia was not used, and it appears doubtful whether there is any reason to use it in patients who have passed the acute stage of bleeding, unless a temporary occlusion of a major artery is indicated during the operation. If, however, early surgery (McKissock's group A) is adopted as a special method for reducing primary mortality, hypothermia seems to be inevitable.[3,17]

In a few cases in this series the aneurysms still filled totally or partially after an incomplete ligature of the aneurysmal neck. However, the long-term results appear to be fairly good, as none of these patients had had any further bleedings. A narrowing of the aneurysmal stalk with a ligature or a clip probably lowers the pressure inside the aneurysm just as much as does a carotid ligature.

A special problem is offered by the large sessile aneurysms at the carotid bifurcation, in which a ligature is not feasible and which are too large for any type of clips. We have wrapped these lesions with muscle, but it is uncertain whether the long-term results are reliable; in connection with other types of aneurysms we have seen secondary ruptures. Here Dutton's[4] method with acrylic plastic should perhaps be tried.

The follow up shows that the fact that the patients had tolerated the

operation well and been discharged from hospital in a satisfactory condition, did not guarantee that they would regain full working capacity. The number of totally disabled was, however, very small and the patients who had made a complete recovery formed a bare majority. The symptoms of which some of the patients with a partial working capacity complained were rather vague and could not always with certainty be considered operative residuals.

SUMMARY

A series of 60 aneurysms of the internal carotid artery and 9 of the carotid bifurcation in 65 patients is presented.

Conservative treatment: 17 patients, 10 of whom died in recurrent hemorrhage.

Carotid ligature: 4 patients; 1 died.

Direct attack: 46 aneurysms in 44 patients; 4 operative deaths. Ligature of the stalk with linen thread or clipping was performed whenever possible. The operative technique is described.

The *follow up* showed that out of the 40 survivors 2 patients had died of operative sequelae. Thus the total mortality was 14 per cent. The present state of the survivors is given.

The advantages of direct attack on aneurysms of this type is stressed.

ADDENDUM

From January 1, 1957 to May 31, 1958, 34 additional aneurysms of the internal carotid artery or its bifurcation have been diagnosed in 33 patients. Eleven aneurysms were not operated upon; 1 patient in this group died in the hospital from recurrent hemorrhage. On 2 patients with infraclinoid aneurysms carotid ligation was performed and well sustained. Of 3 patients whose aneurysms were wrapped with muscle, 1 died suddenly the day after the operation, possibly of heart failure; and 1 died from recurrent hemorrhage 3 months after discharge, which would seem to emphasize the unreliability of this method. Nine aneurysms were clipped, and a further 9 were ligated with thread without mortality; in the former group there was 1 patient with postoperative hemiparesis and in the latter, 1 patient with hemiplegia caused by postoperative thrombosis of the carotid.

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POSITRON-SCANNING WITH COPPER-64 IN THE DIAGNOSIS OF INTRACRANIAL LESIONS

PARTITION OF COPPER-64 VERSENATE IN, AND EXCRETION FROM, THE BODY*

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THE development of apparatus for automatic coincidence scanning for localization of brain tumors by means of a positron-emitting isotope (described by Brownell and Sweet;[3] Sweet and Brownell;[11] Aronow and Brownell[1]) has enabled us to study the suitability of several such isotopes. The technique used and results obtained using Copper-64 in the localization of intracranial lesions will be described and compared with those following the use of Arsenic-74, and the experimentally determined distribution and excretion of Copper-64 both in normal and neoplastic tissues have been studied and constitute the basis of this report.

Wrenn *et al.*[14] employed Copper-64, injected as a phthalocyanate for the localization of brain tumors. In the present series the great majority of patients received Copper-64 as a versenate—Copper-64 ethylene-diamino tetra-acetic acid (EDTA). In a few instances the copper ammonium complex ion has been given as the nitrate $[Cu(NH_3)_4]\ (NO_3)_2$.

METHODS

1. *Preparation and Administration of the Isotope*

Copper-64 can be produced in high specific activity either by cyclotron bombardment (d-p) of copper foil, or by pile irradiation (n-γ) of copper wire.¶ Copper-64 decays, emitting gamma radiation of 1.34 Mev. with 19 per cent β^+ emission: 42 per cent K-capture and 39 per cent β^- emission. Its half life is 12.8 hours. By the time of injection its specific activity is usually less than 1 mc./mg. of copper.

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‡ At present at Médecin des Hopitaux Psychiatriques, Paris, France.

¶ We are now obtaining Copper-64 at very high specific activity from Abbott Laboratories at Oak Ridge produced by a Szilard-Chalmers reaction.

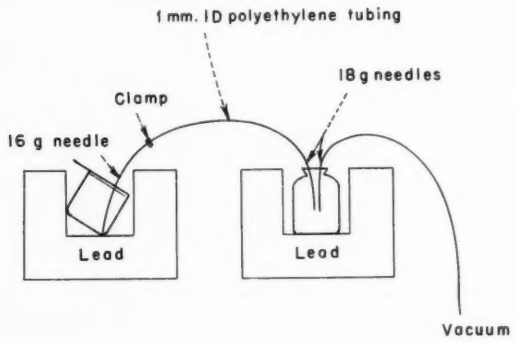


FIG. 1. Copper scheme of H. B. Carter, Jr.

(a) A chelated form of Copper-64 is prepared by dissolving the copper wire in (N/3) nitric acid and adding 10 per cent sodium carbonate until a stable blue precipitate is formed. After filtration the calcium salt of versene (EDTA) is added in excess, at least 100 mg. of versene being used for every mg. of copper. The pH of the solution is about 7.2 after autoclaving and it may then be administered intravenously. The dosage is 2.0 mc. per 70 kg. body weight and this contains about 60 mg. of versenate and 3 mg. of copper. In no case has any untoward effect been noticed even in cases in which this chemical dosage was exceeded by a factor of three.

An alternative and simpler method used only in the latest patients is to dissolve the Copper-64 wire in 6N nitric acid and to adjust the pH of the solution with a mixture of Na_2CO_3—versene. The resulting solution is transferred to a vial and is ready for administration after autoclaving. This method of H. B. Carter, Jr. of the Physics Research Laboratory, Massachusetts General Hospital, is carried out as follows:

Copper Scheme (see Fig. 1).

1. Dissolve copper wire in 3 ml. of 6N HNO_3.

2. Raise pH of solution to 6.5–7. Add 15 ml. of Na_2CO_3 versene mixture (620 ml. of a 10 per cent Na_2CO_3 sol.+380 ml. versene).

3. Transfer solution to vial via vacuum.

(b) Ionic copper is produced by dissolving the irradiated wire in nitric acid and neutralizing with ammonium hydroxide, just enough excess being added to convert the solution to the copper ammonium complex ion, this being necessary to keep the Copper-64 in solution at body pH. The solution is then autoclaved.

2. *Scanning Procedure*

Almost immediately following intravenous injection of the Copper-64 versenate, automatic coincidence scanning was commenced. One must give a greater amount of radioactivity as Copper-64 than as Arsenic-74 to achieve scans of similar density with the two isotopes (the latter is the isotope generally and routinely used in this laboratory). Scans have been carried out at varying intervals after the injection in order to determine the optimal time for the procedure. Although the pictures at 3 hours showed perhaps a little better definition, such a delay in the use of this isotope with its relatively short half life was not practicable in terms of the maximal number of scans with each shipment of Copper-64. In fact, as we shall see later, the

biopsy data also indicate that it is best to scan the patient within the first hour after injection. The majority of the patients in this present study were scanned by the automatic scanning equipment and the results will be described later.

3. *Biological Assays*

The Copper-64 content was determined in 3 groups of specimens: (a) Plasma, erythrocytes and urine were assayed to determine the rate of disappearance of Copper-64 from the intravascular compartment and its recovery in the urine. (b) Biopsy specimens of normal brain, tumor, muscle and bone taken during operation at varying times after injection of Copper-64 were studied to learn how concentrations in these tissues would affect the external scanning, and in particular to find the difference in isotopic uptake between normal brain and neoplastic tissue. This is, of course, the principal determinant of the clinical usefulness of the specific isotope for localization of brain tumors. In each case every effort is made to remove normal and tumor material at operation within the space of a few minutes of each other. When several samples of brain and tumor are to be removed, the initial samples are taken from an area that does not affect the blood supply, and thus the isotopic concentration of later samples. (c) Lastly, 1 case of autopsy material provides data of Copper-64 concentration in the major organs and tissues in the body.

4. *Measurement Techniques*

In each series of determinations of Copper-64, aliquots of a standard solution of the radioisotope given the patient were counted with the samples, thus assuring equal decay of the standards and samples so that computation of the results reflects only biological events. Tissue concentrations throughout are expressed as per cent total dose per kilogram (or liter) of sample normalized to 70 kg. Some of the patients have received two radioisotopes; such double tracer data were analyzed by the method of Michel *et al.*[6,9,12] Activity in the erythrocytes was calculated from the values given by plasma and whole blood by the relationship

$$C_E = \frac{C_B - (1-H)C_P}{H},$$

where C_E, C_B and C_P are the concentrations (expressed as per cent dose/liter) in the erythrocytes, whole blood and plasma respectively, and H is the hematocrit. Samples of urine were similarly assayed for radioactive copper activity and the results were expressed as a percentage of the total dose given.

Samples of organs were counted immediately. After wet weighing on waxed celluloid to minimize losses by drying, the samples were placed on dry ice and rapidly frozen, following which they were transferred to test tubes. Radioactivity was then measured in the gamma well. An appropriate standard was used in all cases. Finally histological aliquots were obtained from the samples of both normal and tumor material and routine staining techniques were employed for microscopical sections.

RESULTS

1. *Scans*

In general, the scans obtained with Copper-64 are similar to those already described elsewhere for Arsenic 74.[11] The normal positrocephalogram (Fig. 2) shows a concentration of isotope in the face area and overlying the tem-

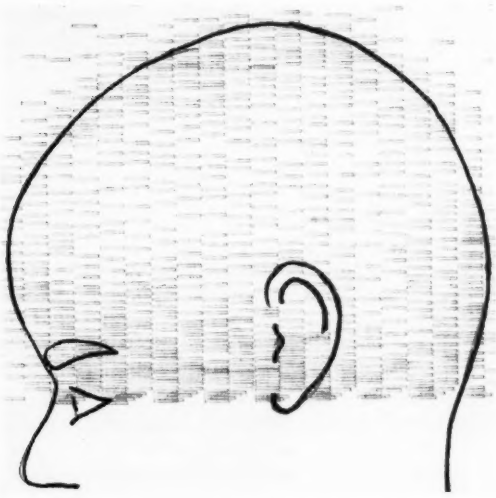


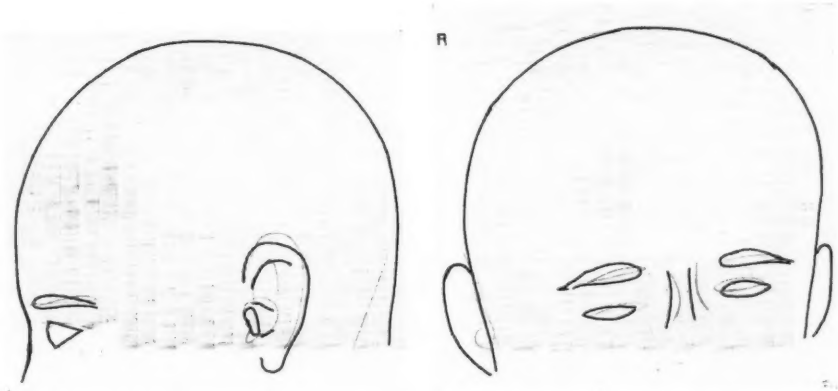
FIG. 2. Positrocephalogram of normal subject. Note increased concentration over face area and over peripheral outline.

poral muscle, and the peripheral outline (scalp plus bone) is well defined. This latter feature is particularly evident in the Copper-64 scans. In addition, the low concentration of Copper-64 in normal brain tissue is another distinguishing feature which may allow a sharper definition of abnormal concentration than is seen in some Arsenic-74 scans. A positrocephalogram on a patient with a brain tumor shows an increased concentration over the area of abnormality. Fig. 3 shows the positrocephalogram on a patient with recurrent astrocytoma. The lateral view shows the increased concentration of isotope in the anterior frontal region. The sagittal view of the same patient (Fig. 4) shows an increased isotopic uptake in the medial zone above the right orbit and nose. The asymmetrogammagram (AGG), not illustrated, showed only slight asymmetry to the right because of the midline position. This favorable position and size were confirmed at operation.

The high uptake of most isotopes by muscle in the temporal and suboccipital regions obscures in these areas diagnostic concentrations recognizable elsewhere in the brain.

Scans of 133 patients have been analyzed; the pictures were diagnosed independently of the clinical findings and were classified as either normal or abnormal. Both unmistakably abnormal and probably abnormal isotopic uptakes were classed as abnormal, whereas the probable as well as the definite normals were all placed in a normal group.

Tumors (Table 1). In this category 37 scans have been reviewed. We were able unequivocally to locate the 4 glioblastomas but missed 3 astrocytomas, grades I and II. Two of these astrocytomas were in the posterior fossa, a



FIGS. 3 and 4. Positrocephalograms of patient with recurrent astrocytoma. (*Left*) Lateral scan. Increased concentration of isotope is in anterior frontal area. (*Right*) Sagittal scan. Increased concentration is in medial zone above orbit and nose. The areas indicated corresponded to position of tumor at operation. (In the original scan the zone of increased concentration stands out in a striking fashion which is unfortunately lost in the reproduction, apparently because of the particular ink used.)

region in which positive isotopic concentrations are difficult to recognize. Cerebral metastases were correctly located in 8 out of 13 cases. The 5 not located were in a cerebral hemisphere.

Four of the 5 meningiomas were correctly localized. The one not recognized was a recurrent lesion of the sphenoidal ridge. When the scan of a

TABLE 1

Results of positron scans of patients with verified intracranial tumors

Tumors	Abnormal Isotopic Concentration	No Abnormal Isotopic Concentration
Glioma		
Glioblastoma multiforme	4	0
Astrocytoma (Grades I and II)	0	3
Oligodendroglioma	0	1
Metastases	8	5
Meningioma	4	1
Acoustic neuroma	0	1
Chromophobe adenoma	1	2
Third ventricle tumor	0	1
Pineal tumor	1	1
(Sarcoma of orbit)	1	0
Melanoma	1	0
Cholesteatoma	0	1
(Osteoma of skull)	0	1
	20 (54%)	17 (46%)

TABLE 2

Results of positron scans of patients with non-neoplastic lesions

Lesion	Abnormal Isotopic Concentration	No Abnormal Isotopic Concentration	Totals
Vascular			
Thrombosis	5	17	22
Aneurysm	0	1	1
A-V malformation	0	2	2
	5 (20%)	20 (80%)	25
Seizures	3 (9%)	29 (91%)	32
Miscellaneous	5	34	39

meningioma is positive, the focal isotopic concentration is usually so striking that the pathological diagnosis can be ventured from the appearance of the scans, especially from the extreme density in the AGG if the tumor is superficial.

As previously mentioned, the accuracy of the procedure is limited in tumors of the posterior fossa. In our 1 case of acoustic neuroma, in which the diagnosis already seemed likely both clinically and by pneumography, we were unable to demonstrate this tumor by scanning. In those cases in which the tumor was supposedly small and near to the midline, as in the region of the 3rd ventricle and sella turcica, the accuracy of the method is also wanting. Three pituitary adenomas were successfully operated upon, but in only 1 case were we able to demonstrate a focal isotopic concentration in this region before operation. Three lesions in the neighborhood of the 3rd ventricle were scanned and 2 of the scans were considered to be normal. Two of these patients subsequently had ventriculocisternostomies carried out for tumors in the pineal region; only 1 of these had an abnormal scan. In the third case a lesion in the 3rd ventricle was suspected, but the patient was discharged when the scan and other studies showed this to be unlikely.

The 1 case of sarcoma was a highly undifferentiated tumor of the lateral wall of the orbit and temporal fossa and therefore was not strictly an intracranial lesion. This patient had several successive scans in the 6 months between the discovery of the lesion and her death and they showed conclusively the progress of the lesion. No abscesses are included in this series. An osteoma of the skull gave a normal scan.

Vascular Lesions (Table 2). In this group, 22 of the patients had possible or proven thromboses and of these only 5 had abnormal scans. The vessels afflicted in these 5 cases were respectively the retinal artery (1 case), the internal carotid artery (1 case) and the middle cerebral artery (2 cases). In the fifth patient, with serious mental deterioration, the site of vascular disease was not determined but the abnormality in the scan corresponded to the hemisphere controlling the weak limbs. In addition to these patients, studies of 3 others, 1 with a capillary angioma questionable even by serial

angiography, 1 with an angioma of the brain stem and 1 with a berry aneurysm of the posterior inferior cerebellar artery all yielded normal scans.

These results therefore support the suggestion made elsewhere in respect to arsenic scans that patients who have tumors are much more likely to show positive scans than those suffering from cerebrovascular conditions. No hematomas are included in this series.

Convulsive Seizures (Table 2). Of 32 patients with seizures of non-neoplastic origin, only 3 had abnormal scans. One had cerebral atrophy; in another, a woman of 35, no abnormality to account for her seizures of 2 years' duration could be seen on pneumoencephalography or arteriography. The electroencephalogram showed moderate abnormality with slow activity in the temporal region, and the scan was decisively abnormal in this area. This patient is still under investigation in the Out-patient Department. The third patient is considered to have had focal ischemic episodes.

The scan, therefore, did not contribute to the diagnosis in 29 cases (90.7 per cent).

Miscellaneous (Table 2). The remaining 39 cases comprise a typical group of patients regularly seen in neurosurgical practice and in whom the diagnosis of brain tumor might be suspected. In 34 the scan was normal. The 5 abnormal scans were in cases in which the diagnosis was 1) cerebral degenerative disease, 2) ? brain tumor but unproven, 3) fractured skull with resultant underlying cerebral atrophy, 4) Paget's disease of the skull, and 5) ? metastases to brain following radical mastectomy for carcinoma of breast. With normal pneumoencephalograms and angiograms and dubious clinical findings in this patient we have as yet hesitated to operate on the basis of the scans alone.

2. *Biopsy Data* (Fig. 5)

(a) Nineteen samples of normal areas of brain from 16 patients were analyzed. Biopsy material was also taken from several other patients; it is not included here because of errors in either sampling procedures or calcula-

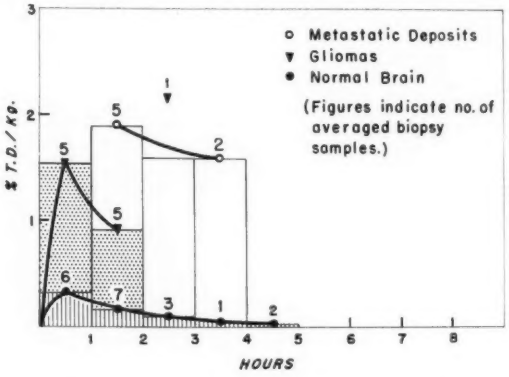


FIG. 5. Concentration of Copper-64 versenate in metastatic deposits, gliomas and normal brain.

tions. In the early period after administration of isotopes, there is a rapidly changing picture so far as the intravascular and intracellular concentrations of the isotope are concerned. The results are therefore more likely after 30 minutes to reflect accurately the state of affairs at the time of scanning. The data are expressed as per cent of dose per kg. (normalized to 70 kg. in each case). The wisdom of examining histological aliquots of the material obtained from operation was shown in 1 case of oligodendroglioma in which biopsies thought macroscopically to be normal brain, subsequently proved on examination of the slides to consist of tumor in from 30 per cent to 95 per cent of the specimen.

The 19 determinations of normal brain have been plotted by hours in order of increasing time after administration of the isotope. The data so arranged indicate that the concentration of copper initially rises to 0.34 per cent TD/kg. within the first hour, falls in the second hour to 0.16 per cent TD/kg. and thereafter declines more gradually.

(b) Copper concentrations have been measured in 18 biopsies from 15 patients with proven brain tumors. In view of the small numbers in this series, it is unprofitable to present the results in graphic form except for those obtained in the first two groups, namely metastases and gliomas—the latter group comprising 3 glioblastomas and 2 astrocytomas. (A wide spread of concentrations is obtained in the biopsy samples and this is explicable not only on the basis of differing types of tumor but also on the wide variations in their vascularity, necrosis, anaplasia and stroma. When, as is usual, several biopsies have been taken of a single tumor, strikingly different concentrations have often been found ranging between that found for normal brain to various higher concentrations in other areas of the tumor.) Two biopsy studies from each of 2 meningiomas revealed 5.2 per cent of total dose per kg. tumor in the patient biopsied less than 1 hour after injection and 1.25 per cent in the patient biopsied 5–6 hours after injection. An acoustic neuroma showed 4.15 per cent at 1 hour and a cerebellar hemangioblastoma 0.75 per cent at 4–5 hours (average of 2 biopsies). Concentration in muscle rises up to 4 hours after injection whereas the concentrations in brain and tumor fall rapidly after the first hour. Consequently the optimal time for the scan is within the first hour after injection.

(c) Copper-64 concentration in 14 biopsy samples of muscle from 14 patients and 13 samples of bony skull from 13 patients are illustrated (Fig. 6). The data again are too meager to analyze critically, but it can be seen that the average concentration in muscle is much higher than in bone, and is in fact greater than that seen with some gliomas, so that it is not surprising that the temporal and nuchal muscles obscure tumors beneath them.

3. *Autopsy*

Only 1 patient has come to autopsy within the physical decay period of Copper-64, which had been given 19 hours prior to death. Samples from various organs and tissues were obtained post mortem within a few hours

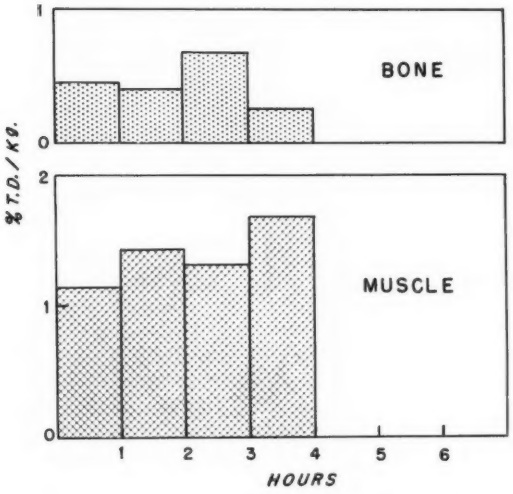


FIG. 6. Concentration of Copper-64 versenate in bone and muscle.

of demise and were measured immediately for radioactivity as previously described. Table 3 shows the various concentrations found in the organs listed in this one patient, who died from a secondary carcinomatous deposit in the left temporoparietal region.

TABLE 3

Showing concentrations in various organs of the body (Copper-64 had been given 19 hours prior to death)

No.	Tissue	Weight of Tissue (Gm.)	% Total Dose/ kg./"N"*
1	Liver	0.8568	30.20
2	Pancreas	1.3842	9.02
3	Kidney	1.0847	7.78
4	Tumor†	0.4279	4.06
5	Tumor†	0.8435	3.31
6	Skull bone	0.5370	3.14
7	Lung	0.8901	2.81
8	Spleen	1.1368	2.29
9	Aorta	0.9136	2.15
10	Lumbar vertebra	1.0916	2.08
11	Heart muscle	1.1784	1.42
12	Temporalis muscle	1.2824	0.93
13	Cerebellum	1.0636	0.15
14	Cerebral (gray matter)	0.6045	0.14
15	Cerebral (mixed)	1.2665	0.13
16	Cerebral (white matter)	0.5359	0.08

* Normalized to 70 kilo.

† Secondary carcinomatous deposit in left temporoparietal region.

From Table 3 it can be seen that the chief sites of Copper-64 concentration are in the liver, pancreas and kidney. At 19 hours 30.2 per cent/kg. of the total dose given is found in the liver followed by pancreas, 9.02 per cent and kidney, 7.78 per cent. Lung, spleen and bone (skull and lumbar vertebra) each contained between 2–3 per cent. One sample of tumor removed from the brain at autopsy contained 4.06 per cent/kg. of the total dose administered (another sample contained 3.31 per cent) compared with an average of 0.12 per cent in normal brain, giving a tumor-brain ratio of between 34 and 27.

4. *Concentration in Plasma and Erythrocytes*

The time-course of concentration of Copper-64 versenate in plasma and erythrocytes has been obtained in 4 patients in normal health. (In the 3 patients in whom the intravenous injection took 5–6 minutes, the average for all determinations in plasma and the averages in whole blood and erythrocytes are recorded in Fig. 7. In the fourth case the injection took 14 minutes, and in the fifth, plasma values alone were obtained.) The peak of concentration in plasma and erythrocytes is maximal almost immediately after the completion of the injection, declining rapidly thereafter during the remainder of the first hour and then remaining fairly level in all components for the next 3 hours.

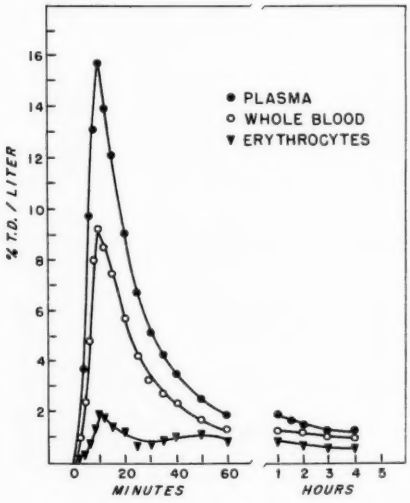


FIG. 7. Concentration of Copper-64 versenate in plasma, whole blood and erythrocytes, showing average in 3 patients.

5. *Excretion in Urine and Feces*

In each of the 5 normal patients, the urinary excretion of Copper-64 was determined. This is illustrated in Fig. 8 in which the ordinate records cumulative excretion as a per cent of the injected dose. It will be noted that the major proportion of the early excretion is contained in the first specimen (2–6 per cent of total dose) and it is clear that nearly all of the copper is bound in a form in which excretion is extremely slow. Because of the short half life of Copper-64 and the natural delay in obtaining fecal specimens, it has not been found possible to measure excretion by this route.

It is, therefore, assumed that about 5 per cent of the total dose is excreted via the urinary tract and that 95 per cent of each dose of Copper-64 is retained in the body until its activity ceases by physical decay.

6. *Radiation Effects*

On the basis of the foregoing data we have calculated the radiation dose received by the patient assuming that 95 per cent of the injected dose is retained and about 5 per cent excreted in the urine. Approximately 30 per cent of the copper is found in the liver and the remainder of the activity is as-

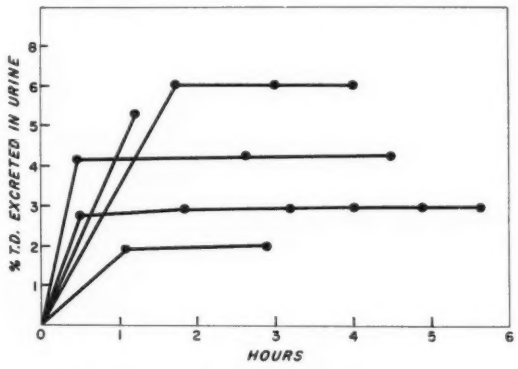


FIG. 8. Urinary excretion of Copper-64 versenate.

sumed to be distributed uniformly throughout the rest of the body. Whole-body and liver radiation doses are calculated separately.[13] The whole-body β radiation absorbed dose in rads, D_β, may be calculated by the equation $D_\beta = 73.8 \times \overline{E} \times C \times T$ in which $\overline{E}$ is the weighted average energy of the beta-ray spectrum, C is the concentration in μc./gm. and T is the effective half life in days, which is equal to the physical half life in this case. The gamma-ray whole-body radiation dose is similarly calculated from the equation $D_\gamma = 1.44 \times \Gamma \times C \times G \times T$ in which Γ is the gamma-ray dose-rate constant and is 7.5 cm^2-r/mc-hr for Copper-64. G is the geometrical factor and C and T have been previously defined.[8]

The radiation doses received by a patient following an injection of 2 mc. of Copper-64 are as follows:

	Total Beta	*Total Gamma*	*Totals*	
Whole body	0.065	0.26	0.325	rads
Liver	1.29	1.89	3.18	

Positron, β^+, and negatron, β^-, emission are included under the heading of beta dose while gamma radiation consists of primary gamma emission as well as annihilation radiation.

It is seen that the total whole-body radiation is very low but that the liver receives locally 3.18 rad. This is felt to be a comparatively small dose to this organ (see discussion below).

DISCUSSION

In 1951 Sweet[10] as well as Wrenn and co-workers[14] pointed out the value of the positron method of coincidence counting and its advantages over single channel scanning utilizing gamma radiation. In 1952 Brownell[3] designed the apparatus to carry out continuous automatic coincidence scanning, and in 1955 Sweet and Brownell[11] discussed the choice of the ideal isotope and gave reasons for their selection of Arsenic-74. Recently further improvements have been made in the engineering and electronic design of the machine.

Copper[4] is found in the normal human adult body in small amounts (100–150 mg.)—circa 64 mg. being found in muscle, while bone and liver contain about 23 and 18 mg. respectively. The remainder is evenly distributed in the body tissues. The chief channel of excretion is via the intestinal tract, both directly through the intestinal wall and in the bile. Further small amounts are lost in the urine, in the normal menstrual flow and in the sweat. The highest concentration is found in the liver. In the blood, copper is distributed approximately equally between the erythrocytes and the plasma. In plasma 90 per cent (if not all) of the copper is contained in the complex protein molecule—caeruloplasmin which has been identified as an α_2 globulin.[2] Copper also has a great affinity for porphyrins and is probably excreted from the liver and in bile as copper bilirubinate.

Experimentally in dogs and rats,[5] oral administration of Copper-64 causes a rapid rise of Copper-64 in the blood plasma and later in the erythrocytes. Liver, kidney and bone marrow accumulate most of the Copper-64. Excretion is 75 per cent via the intestinal tract but only 5 per cent is found in the urine after 5 days. Intravenous administration, however, is approximately 10 times more effective in terms of concentration of Copper-64 in the tissues and a maximum of 6 per cent is lost in the urine.

Copper-64 has the advantages that it is an inexpensive reactor-produced isotope and it is readily and easily available. Copper-64's main disadvantage of a relatively short half life of 12.8 hours coupled with the fact that only one shipment per week has been possible from the Brookhaven reactor accounts for the small numbers presented in this preliminary study. However, these disadvantages may be overcome by the proximity of a nuclear reactor to the hospital. Even so, it is possible with the usual shipment of 50 mg. of Copper-64 to produce enough material to enable 5–7 scans to be carried out in the 24 hours following delivery. Much useful information has been collected concerning the relative and absolute values of both Copper-64 and Arsenic-74 by arranging the admission of patients to the hospital in such a way that the Copper-64 scan may be followed by an Arsenic-74 scan. At subsequent operation biopsies were taken and analyzed in respect to the concentrations of both of these isotopes in the tissues.

Attempts to gain further data by continuous counting *in vivo* with Copper-64 by means of probes in the brain have been disappointing because the low energy of its beta particles and a substantial gamma-ray emission give

an insignificant difference in the counting rates from probes in brain and tumor respectively.

The low total-body dose of radiation from Copper-64 is a further distinction from other isotopes. This is of the order of the weekly permissible dose for personnel constantly exposed to radiation. The high liver concentration may be used to advantage and considerable success has been achieved with liver scans. It is hoped that neoplastic concentrations may be visualized by this technique as areas of low uptake in the denser liver shadow. The relatively low radiation even to the liver makes possible repeated scanning with this isotope.

In mice, the toxicity of copper is high but in man no harm has ensued even when single large doses up to 9 mg. of copper have been given. However, it is imperative that high specific activities be used in order to avoid large amounts of chemical copper.

At present it has not been found possible to estimate quantitatively the ionic and chelated copper in micro amounts in the blood, and we have no means of knowing how long Copper-64 remains in its chelated form after injection. In those cases in which Copper-64 was injected for the purpose of scanning initially in the ionic form and later in the same patient as the chelate, better definition was achieved with the latter. The evidence so far available shows that the urinary excretion of Copper-64 after administration of the chelate is about 10 times greater than after ionic copper; moreover in dogs there is a greater uptake of ionic copper than of chelated copper by the liver. These facts suggest that chelated copper remains in a more discrete and mobile form after injection than does the ionic form.

Locksley *et al.*[7] have shown in mice that tumor tissue tenaciously holds onto copper while the concentration in the brain falls off (in a manner parallel to other tissues). Hence in mice the tumor/brain ratio increases with time. The relatively low concentration of Copper-64 in brain tissue of the mouse was also pointed out. In the present series our biopsy data are too scanty to enable us to be dogmatic, although we also have found a low concentration in normal brain—0.16 per cent total dose/kg. at 1–2 hours for Copper-64 as compared with 0.25 per cent at this time for Arsenic-74. But the tumor/brain ratios were not as high in man as Locksley *et al.* found in mice.

Seventy patients have had both Copper-64 and Arsenic-74 scans in which the two isotopes were given within days of each other, and the results are, therefore, comparable in that approximately similar conditions obtained with either isotope. In those with proven tumors (Table 4A), an abnormal isotopic concentration in the area of the tumor was present in 55 per cent of cases with Copper-64 as compared with a 76 per cent accuracy with Arsenic-74. In 20 tumors the scans gave roughly similar results with each of the two isotopes; in 7 the arsenic scan showed the tumor much more clearly than the copper scan; in 2 the reverse was true, the copper picture being much clearer.

Fifteen vascular lesions (Table 4B) were scanned with both isotopes and

TABLE 4

Comparison between Copper-64 and Arsenic-74 scans in the same patient

A. VERIFIED NEOPLASMS

Tumor	Abnormal Isotopic Concentration Seen with Copper-64	Abnormal Isotopic Concentration Seen with Arsenic-74	Totals
Glioma	4	8	10
Metastatic tumor	6	6	8
Meningioma	4	5	5
Acoustic neuroma	0	0	1
Pituitary tumor	0	0	1
Pineal tumor	1	1	2
Third ventricle tumor	0	1	1
Sarcoma of orbit	1	1	1
	16 (55%)	22 (76%)	29

B. NON-NEOPLASTIC DISORDERS

Lesion	Abnormal Scan		"Suspicious" Scan		Normal Scan	
	Cu-64	As-74	Cu-64	As-74	Cu-64	As-74
Vascular						
Thrombosis, hemorrhage or aneurysm	2 (13%)	4 (26%)	2	1	11	10
Seizures	0	0	3	6	16	13
Miscellaneous	2	3	2	4	12	9

it may be seen that in the abnormal group only 2 copper scans as compared with 4 arsenic scans were considered to show focal concentrations of the isotope. Two copper scans and 1 arsenic scan were designated as "suspicious" of abnormality, while in the normal group there were 11 copper scans and 10 arsenic scans. Scans localizing 3 of 18 lesions appeared in an earlier series with Arsenic-74 in which the patient's subsequent progress pointed to a diagnosis of cerebral thrombosis and/or hemorrhage. However, in this series, of 15 lesions diagnosed as cerebral vascular thrombosis or hemorrhage, the Copper-64 scans were positive in only 2 of these (13 per cent) as compared with 4 (26 per cent) in the case of Arsenic-74. These later data intimate that there may be less chance of a misleading result if Copper-64 is used in those cases in which the diagnosis lies between a cerebral neoplasm or vascular accident.

In the seizure group, 19 patients were scanned with both isotopes. None of the scans was considered to be abnormal with either Copper-64 or Arsenic-74 but only 3 copper scans were classified as suspicious compared with 6 arsenic scans. Sixteen copper and 13 arsenic scans were normal.

In the miscellaneous group, 17 patients were scanned. With Copper-64 12 were normal, that is 70 per cent accuracy as compared with a 51 per cent accuracy with Arsenic-74.

Upon comparing Copper-64 with Arsenic-74 for scanning we conclude that: the physical advantage of a low total-body irradiation to the patient is greater with Copper-64 than is the case with Arsenic-74. Clinically Arsenic-74 has localized substantially more tumors. However, Copper-64 scans have been positive in some patients with neoplasm when the Arsenic-74 scan was negative although the reverse has more often been true. When the differential diagnosis lay between neoplasm and a vascular accident, copper scans have shown an advantage over arsenic in that they were less likely to yield a false positive. Hence we are continuing the intensive use of both isotopes.

SUMMARY AND CONCLUSIONS

1) Copper versenate injected intravenously in man achieves within 2 hours concentrations in metastatic tumors about 9 times, and in more rapidly growing gliomas about 5 times those in normal brain. Concentrations of copper in muscle rise steadily whereas those in tumor and normal brain fall.

2) Copper scanning, best carried out within an hour of injection, has detected about 55 per cent of a random series of intracranial tumors. In the same patients arsenic scans detected 76 per cent of the tumors. However, some tumors were detected by copper in which the arsenic failed.

3) Vascular lesions of the brain are less likely to yield a positive scan with copper than with arsenic.

4) About 5 per cent of an intravenously injected dose of copper versenate is excreted in the urine. The rest largely undergoes physical decay of its activity before excretion occurs.

5) The Copper-64 activity injected for a diagnostic scan gives a whole-body radiation dosage of 0.33 rad., which is about the weekly permissible dose for personnel constantly exposed to radiation. Hence this diagnostic maneuver may be repeated several times with impunity.

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SURGICAL RECONSTRUCTION OF OCCLUDED CERVICAL CAROTID ARTERY

REPORT OF A SUCCESSFUL CASE WITH 4-YEAR FOLLOW UP AND THREE EXAMPLES WITHOUT SUCH TREATMENT*

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THE clinical syndrome of occlusive disease of the carotid arterial system has been well described.[4–7,11] Predilection for arteriosclerotic thickening of the wall of the vessel and formation of plaques has been shown to be at the bifurcation of the common carotid artery and on up into the internal trunk.[1,12] Thrombotic disease of the carotid tree cephalad from the aortic arch, including temporal and ophthalmic branches, has also been attributed in rare instances to infection or trauma.[14] Narrowing of the lumen can advance so insidiously that recurrent and progressive neurologic deficit may be overlooked or not ascribed to decrements of carotid-cerebral blood flow. Successful direct treatment of the abnormality depends on its prompt recognition and evaluation to ascertain the possible value of surgical reconstruction of the affected artery.

CASE REPORTS

Case 1. A 64-year-old man (right-handed) was admitted to hospital on Feb. 21, 1950, having experienced drooping of the right side of the mouth and weakness of both legs 8 years previously, lasting 8–9 months. He had had a fainting spell 5 years back with loss of consciousness. Three weeks before hospitalization he became drowsy and his tongue and mouth pulled to the left, clearing rapidly. One week before admission the left arm became weak and vision began to fail, beginning with diplopia and declining to perception of light and gross movements of the hand within 3–4 days. (The sketchy history probably omitted other interval episodes of neurological difficulty.)

Examination. There was poor comprehension because of difficulty in language. The patient was mentally dull but oriented and able to perform simple commands. He could not read letters 4 inches high but named a few objects adequately visualized. There were nystagmoid movements of the eyes on lateral gaze; pupils and fundi were normal; fields of vision revealed large central scotomata surrounding points of fixation and extending beyond blind spots. There was weakness of the left lower face and left arm, with slight increase in deep reflexes generally; plantar reflexes were equivocal; sensation was grossly intact. Blood pressure was 220/100.

Laboratory Studies. Hemoglobin was 16 gm. per cent, hematocrit 50 per cent, and count of white blood cells 8,800/c.mm. Cerebrospinal fluid was slightly xantho-

* Read before the American Academy of Neurological Surgery, Sea Island, Georgia, November 13, 1957.

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chromic; protein was 60–80 mg. per cent. Hinton was negative, blood urea nitrogen 16 mg. per cent, and alkaline phosphatase 3.5 K.A. units (normal). Electrocardiogram showed frequent premature beats and indication of hypertension. Roentgenograms of skull and chest were unremarkable.

Course. Bilateral percutaneous carotid arteriography was carried out at one stage using 35 per cent Diodrast. Upon injection of the left carotid the anteroposterior view showed the internal carotid dividing into its anterior and middle branches as well as the contralateral anterior and middle branches filling via the anterior communicating artery, all vessels being in normal relationship. Upon injection of the right side there was no filling of the internal carotid in both anteroposterior and lateral projections although the needle was shown well placed in the common trunk with filling of the external carotid system. Following arteriography pupillary inequality developed and the patient was responsive only to painful stimuli. Subsequent spinal puncture revealed a pressure of 140 mm. and slightly yellow cerebrospinal fluid. He remained comatose and expired 3 days later.

Autopsy disclosed advanced atherosclerosis involving all major cerebral arteries. There was extensive and complete obstruction of the right internal carotid but the anterior and middle cerebral arteries on both sides were open; both posterior communicating arteries were extremely narrowed and questionably patent. The brain showed fresh thrombotic occlusion obstructing the full length of a tortuous basilar artery, superimposed on arteriosclerotic plaques with patchy chronic ischemic infarction and acute softening in both occipital and both temporal lobes, thalami, pons and cerebellum. Multiple old infarcts with cystic degeneration were noted in the internal capsule, putamen, and globus pallidus on each side. Generalized arteriosclerosis was widespread in major arteries elsewhere.

Comment. The brain of this man had achieved remarkable toleration to progressive occlusion of the right carotid artery in the face of extensive cerebral arteriosclerosis by the development of compensatory supply through the contralateral circle of Willis and probably meningeal anastomoses[13,15] and the homolateral ophthalmic artery as well.[9] Cerebrovascular survival during the last 3 days of life apparently was maintained almost entirely by the left carotid artery. The extensive old capsular and lenticular lesions bilaterally came as a surprise in view of the relatively minor degree of neurologic loss recorded during his hospitalization. Basilar narrowing was undoubtedly in progress for several years before it was rendered complete by ill-conceived carotid arteriography. Competent observers, including several neurologists and neurosurgeons and two ophthalmologists, suspected a prechiasmal or retrobulbar lesion, perchance a neoplasm of the middle fossa. The possibility of occlusive carotid or cerebrovascular disease was not mentioned by any one of them; apparently no one chose to palpate the carotids in the neck. In retrospect, absent pulsation might have been noted on the right side. Two-stage angiography should then have been performed at 24-hour interval using the less irritating Thorotrast; or surgical exploration of the right cervical carotid system might have disclosed the lesion and sufficient healthy subcranial segment of internal carotid artery available above the site of thrombosis to permit reconstruction. We suggest that such an at-

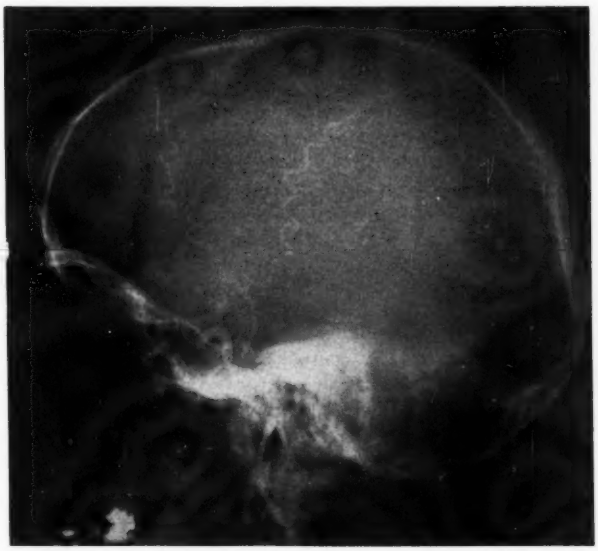

FIG. 1. *Case 2.* Arteriogram shows no filling of left internal carotid above the siphon.

tempt would have been warranted and that its success might have augmented the marginal cerebral circulation in this particular patient.

Case 2. A 46-year-old machine tool worker got up one night in December 1953 to void and became aware of left-sided headache and right-sided weakness. He had to lean on furniture and noted blurred vision in the left eye. He managed to get back to bed. The next morning he was free of weakness but had mild left-sided headache for 12 hours. Four days later, he again awoke with faint left-sided headache and blurred vision. His right side was weak, with a limping gait. He tried to drive his car to work but collided against the garage door to his right. He returned home to bed and improved during the next hours, but was admitted to hospital the same day, where left open carotid arteriography was performed.

Examination. Details concerning history were vague. Cranial nerves were normal. Visual fields were full to confrontation. Grip of the right hand was weak, with drifting of outstretched arm. Left carotid pulse seemed less forceful than right. Blood pressure was 110/75.

Laboratory studies revealed normal clotting time, normal electrocardiogram, negative Hinton, normal hemogram, and normal urine. Fasting blood sugar was 90, nonprotein nitrogen 28 and cholesterol 252.

Diagnosis. Clot in left internal carotid.

Course. Patient was observed closely for recurrence of neurologic disturbance. Arteriogram was considered to show no filling of the left internal carotid above the siphon (Fig. 1). The treatment contemplated was intracranial approach under hypothermia, with removal of thrombus by direct incision of artery. If hemiplegia did not recur for several days the clot might have become organized and adherent so that thrombectomy or resection and graft might be necessary.

This man recovered without recurrence of neurological deficit. No anticoagulants or other medication were administered. He returned to work and has remained well up to the most recent follow up (December 1957).

Case 3. A 48-year-old man (right-handed) began to have occipital headaches early in 1951 and several months later experienced episodes of blurred vision and occasional blindness in the left eye, each lasting for 5–10 minutes over a period of 3 weeks. Two days before hospitalization on April 23, 1951 weakness of the right arm and leg caused him to fall while getting out of bed and he was aware of difficulty in talking and visual loss in the left eye. Symptoms improved during the next 48 hours but his wife noted a drooping of his right lower face, incoherent speech, and that he unknowingly burned the middle finger of his right hand.

Examination. Pupils, visual fields on confrontation, and fundi appeared normal. Left carotid pulsation was poor by palpation and absent above the bifurcation. There was weakness of right arm and leg with hypalgesia and hypesthesia over the dorsal aspect of the right hand and fingers 3-4-5, fading out over the forearm. Plantar response on the right was extensor. Blood pressure was 150/110.

Laboratory Studies. Hemoglobin was 15.6 gm. per cent. Count of white blood cells was 14,000, with 81 polymorphonuclear leucocytes and 16 lymphocytes. Blood glucose was 93 and urea nitrogen 11 mg. per cent. Hinton was negative. Spinal fluid pressure was 150 mm.; protein 137 mg. per cent. Electrocardiogram was nor-

FIG. 2. *Case 3.* Arrows point to irregular narrow carotid artery in neck partially obscured by larger external carotid.

mal. Electroencephalogram displayed left temporal slow waves of low voltage. Roentgenograms of skull were not remarkable.

Course. Percutaneous needling of the left common carotid was not successful. The vessel was exposed and required three punctures to obtain a satisfactory back flow prior to injection of contrast medium (Fig. 2) which showed beaded narrowing of the internal trunk up to its siphon and poor filling of the anterior and middle cerebral branches with normal caliber and filling of the external carotid. Irregular narrowing of the internal carotid artery extended from the bifurcation to its cranial entrance. The sclerotic condition of the artery was also demonstrated by surgical exploration, thus definitely excluding the feasibility of any reconstructive procedure. Cervical sympathectomy was performed, creating homolateral ptosis and miosis. No anticoagulants were given.

This man has been working in manufacture of jewelry for over 6 years with no recurrence of focal neurologic deficit (last follow up November 1957).

Case 4. A 50-year-old widow (right-handed) had periodic near-blackout spells for 8 weeks since March 1953, consisting of feeling of faintness and dim vision for 1–2 minutes, clearing if she bent her head forward; she also experienced unrelated intermittent numbness of the 3rd, 4th and 5th fingers of the right hand. On the day of stroke (May 2, 1953) she tended to doze while driving and that evening after supper suddenly became unable to speak clearly and drooping of the right lower face developed with paralysis of right arm and leg. She was hospitalized for 4 weeks under a diagnosis of cerebral thrombosis during which time improvement became manifest by slight return of movement in the right arm and leg and vocalization of a few syllables.

Examination (June 10, 1953). There was no spontaneous speech and she was unable to understand written words but could repeat a few words upon dictation, and perform addition and subtraction of single digits and indicate the use of common objects. There was indefinite right lower quadrantopsia by confrontation. The right lower face was weak. She could move her right shoulder but no other parts of right upper limb; she was able to elevate right leg 14″ from bed, but there was no motion of foot or toes. There was early spasticity of right arm and leg and atrophy of the musculature of the hand. Pinprick evoked less response over right limbs than left; localization of touch and graphesthesia were absent on right hand, poor on forearm, and fair on leg. Sense of pressure was impaired in right forearm and hand. Tendon reflexes were hyperactive on right with ankle clonus and extensor plantar response.

Course. Five weeks after the ictus open left internal carotid arteriography (Fig. 3A) was performed above the bifurcation which was found to be firm and sclerotic on palpation.

Operation. Surgical reconstruction of the left internal carotid was performed 2 days later (June 11, 1953) under local and intravenous barbiturate anesthesia. The bifurcation was resected and found to be narrowed to a whorl-shaped pinpoint opening by an arteriosclerotic plaque (Fig. 4). The external carotid was ligated and the proximal end of the common trunk was sutured to the tortuous and adequately sound remainder of the internal carotid.

Postoperative Course. It was planned to maintain clotting time at 2–3 times normal, but by error, 20,000 units of high potency heparin were administered intravenously after operation (2 cc. of a 10,000 μ./cc. preparation or 10 times the usual 1,000 μ./cc. dosage). Clotting time was 60 minutes 5 hours later, 12 minutes at

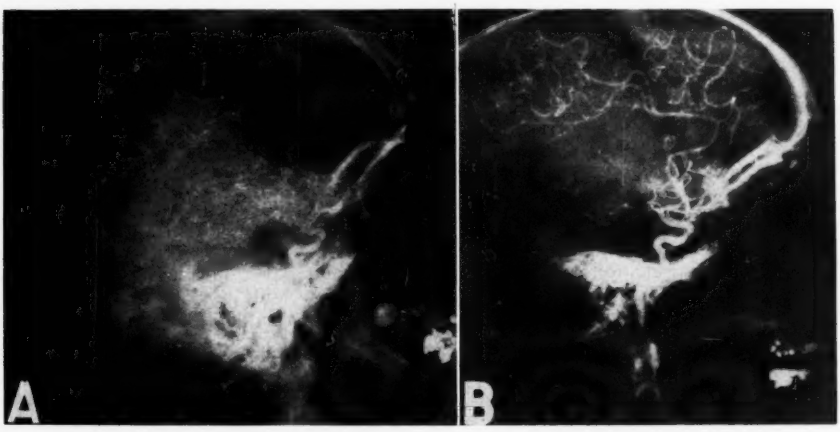

FIG. 3. *Case 4.* (A) Preoperative open internal carotid angiogram. Injection, even though rostral to site of occlusion at bifurcation of common carotid, reveals only thready filling of middle cerebral branches. (B) Five months after reconstruction. Open common carotid angiography—injection 2 cm. above clavicle. In lateral view there is excellent anterior cerebral filling but middle cerebral branches are still poorly outlined. Identical technique was used for both arteriograms.

$8\frac{1}{2}$ hours and 4 minutes at 12 hours; at this time the customary intravenous dose of 2,000 units of heparin was administered and repeated after 2 hours. Approximately 15 hours later a clinical observer noted, "Patient speaks distinctly—moves

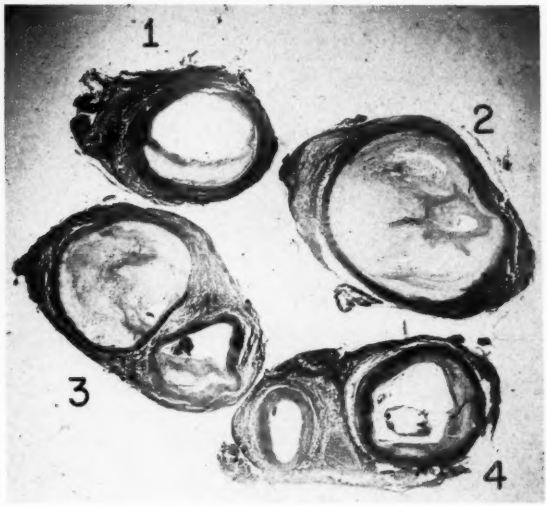

FIG. 4. *Case 4.* Serial transverse sections of resected carotid artery showing degree of arteriosclerotic closure at several levels: (1) common trunk; (2) residual pinpoint lumen at birfurcation; (3) occlusion of internal is almost complete 2 cm. above bifurcation; (4) at 4 cm. both internal and external are patent.

right lower face, not right arm—flexes and extends right thigh actively." Clotting times were maintained between 4 and 55 minutes for the next 6 days by giving 145,000 units of depot heparin distributed through 5 doses as indicated. Two days later flexion was noted for the first time at the right elbow. Ten days postoperatively she was able to name the days of the week and to pronounce her name after a long delay; anomia was still severe but she could select the correct name for an object among a set of choices presented verbally. Five months following carotid reconstruction open arteriography revealed a minimal constriction at the site of the anastomosis (Fig. 5) and a pattern of cerebral vascularity (Fig. 3B) that appeared to represent improvement of collateral supply over the preoperative condition (Fig. 3A). By this time the patient was walking without manual or mechanical assistance and was making consistent improvement in speech and other related mental functions.

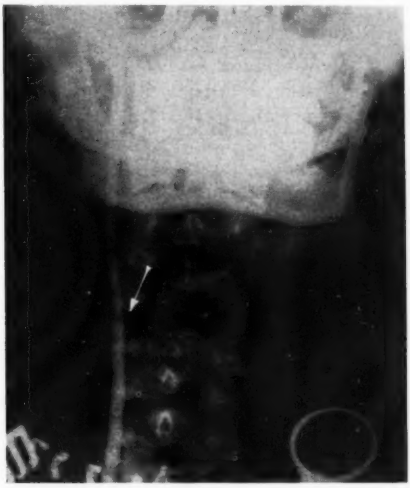

FIG. 5. *Case 4.* In anteroposterior view of neck anastomosis is functioning well. Arrow points to site of suture.

Examination (Jan. 14, 1957). Patient appeared to be in good health and was alert and clearly oriented. Pulsation of left carotid was palpable throughout its cervical course. She understood what was said to her and cooperated well with examiner. She comprehended printed and written words and sentences of ordinary content, and recognized pictures of objects and scenes with respect to identity, color, and meaning. Speech difficulty appeared to be mainly motor. Subject carried out directions with respect to parts of body, right and left and spatial relationship; identified common objects presented to sight and touch despite limitation in ideational and nominal speech. She is said to have a vocabulary of several hundred words—recited serial figures, months of the year, days of the week, simple poetic verses and a few books of the Bible. She repeated words and short phrases on dictation but with hesitation and frequent errors; reading aloud was also confined to short words and phrases. She learned to write with her left hand and produced words and statements, such as name and address and similar remarks, spontaneously and by dictation. Easy arithmetic was performed in both verbal and written answers to problems, but she was unable to subtract serial 7s or to perform multiplication. She would scan a newspaper or magazine page with considerable interest, then pass it to someone to read aloud, the content of which she understood and enjoyed. She indicated that improvement in her mental faculties has continued over the past $4\frac{1}{2}$ years and has been enhanced by coaching from her relatives and by radio and television.

Neurologic Examination. Functions of the cranial nerves were good. The optic fundi appeared normal and visual fields were full by confrontation. Slight asymmetry of face was more noticeable during emotional expression. Right arm was maintained in typical posture of hemiplegic flexion—especially thumb, fingers, and

wrist; there was paralysis of hand and fingers but a fair range of active motion at elbow and shoulder. Right leg was moderately spastic but circumduction gait was remarkably efficient and she avoided scuffing; reflexes were hyperactive in right limbs with pyramidal tract signs. Mistakes were noted in testing sense of distal position, recognition of objects and graphesthesia.

Cortical Function Test (Feb. 4, 1958). Testing done with this 54-year-old woman indicated adequate comprehension of spoken language and good comprehension and memory for nonlanguage visual materials. Comprehension of reading was somewhat impaired. The most marked impairment was inability to express ideas in speech. There was some dysarthria. Writing was also difficult for her. Her attitude throughout the testing was favorable—i.e., she was most cooperative, tried persistently, and did not become disturbed by her difficulties.

On the aphasia test she was often hesitant and slow about evoking words, and when she did so, there were likely to be mispronunciations. For example when asked to evoke words spontaneously, she depended upon things she could see or touch, and there were some substitutions of sound in the names she evoked. She was able to evoke words to complete sentences fairly well and could do most of the automatic word series but with occasional mispronunciations. However, there were a few omissions in the alphabet and in her recitation of a rhyme there were some mispronunciations. In repetitive speech tests she had some trouble with articulation. Also she omitted part of the longest sentence. She was able to evoke opposites to about half the stimulus words given, both orally and in print. Having the stimulus word supplied in a sentence for association helped her occasionally.

Although she was not able to use words well enough to make simple explanations she could use some words without the connectives (e.g., her reply to the question: "How do you make a sandwich?" was "meat, butter, and bread") and she was able to do some naming. She named the common objects with some hesitation and named most of the colors correctly. Also she named letters and some numbers. For the geometrical forms she gave either a description ("round" for "circle") or an association ("the sky" for a star) or a concrete term ("a box" for the square). Although she readily identified both her own and examiner's hands and fingers as examiner gave the names, patient was not able to supply very many of the names herself. Once she gave an association—for the right middle finger she said "sew with" meaning that that is the finger on which the thimble is worn.

She demonstrated her recognition of simple sounds in a test of auditory agnosia, but she could not name these except for coughing. Tactile recognition could not be tested in the right hand, but in the left hand it was adequate except for two coins, both of which she identified as a button.

Comprehension of spoken language was good for single words (e.g., pointing to objects as examiner named them), and for simple questions, but was not as good for the more difficult material of the absurdities. She followed single and twofold oral commands readily and did one of the threefold commands. However, on the other, she did only two commissions correctly. One of these was awkward and slow to perform with her left hand and that probably contributed to her forgetting the third.

Comprehension of printed language was accurate for single words (e.g., matching objects and colors with their printed names) and for single and twofold printed commands. She even did two of the more complex commands correctly, but was a

little insecure about one of these. On questioning she indicated that she reads only the newspaper and that she has trouble seeing ordinary print but that she could see the print used here.

She attempted all writing with her left hand. She wrote her name legibly and was also able to write the date correctly. There were misspellings in the names of common objects and colors. She at first said it was "no use" to try propositional writing, but when urged to attempt a sentence about the weather she said orally, "air, the sun shining, clouds," but was only able to write "sir clow." From dictation she wrote single letters correctly except for substituting *z* for *v*. She wrote primer words correctly but could not write sentences from dictation. She copied correctly from print.

On the nonverbal scale of the Wechsler-Bellevue, she was quick to grasp the various tasks and, even though she had to use only the nonpreferred left hand, she was able to obtain an average score. This indicates good retention of mental ability when language is not involved. Her drawings of designs from memory indicated very good visual memory. Auditory memory was more limited. She was able to do only the simplest problems in mental arithmetic, but she could do written arithmetic quite accurately. Even though orally she once named the wrong process, she carried out the correct one. Ability to spell words orally was quite limited. Her oral reading was done in telegraphic style. She seemed to be reading ahead to get the gist of the material and this may help account for some of the omissions. There were also some substitutions of words.

Comment. The most impressive and indeed crucial improvement in this patient following reconstruction of the carotid artery was in the domain of comprehension and expression of language. The promptness with which betterment succeeded operation and the degree to which it progressed constitute the principal basis for our conclusion that the procedure was beneficial. The postoperative arteriogram (Fig. 3B) demonstrates that augmented nutrition of the language areas had been derived largely from collaterals of anterior cerebral vascular supply. Moreover, the extent of clinical improvement suggests that occlusive disease in the territory of the middle cerebral artery should not deter surgical effort to restore the main truncal arterial supply.

DISCUSSION

The fact that several major arteries supplying the brain are affected by arteriosclerosis, which is exemplified by Case 1, ought not to preclude surgery on a reparable stenosis of the internal carotid, but should rather hasten such procedure lest vital collateral supply be lost by irreversible progress of the disease. The capacity for cerebral vascularity to improve in the face of serious carotid sclerosis is demonstrated by Case 3 and brings to mind the thought that even a temporarily functioning graft might tide the brain over such a crucial period while collateral supply is evolving.[13,15]

Thrombo-endarterectomy alone or resection of a solidly blocked segment of carotid artery has seldom been proven beneficial in cases of complete obstruction; but timely replacement by graft or by-pass installation would

seem to be a promising procedure.[2,10] Anastomosis of a distally patent segment of internal to external carotid artery has been tried with equivocal results.[16] Successful renewal by autogenous vein graft has been described in cases of occlusion or threatened occlusion.[8] However, the ultimate functional durability of any type of graft cannot yet be predicted.*

A case similar to our Case 4 has been recorded by Eastcott *et al.*[3] who relieved a patient from attacks of hemiplegia for 5 months up to the time of their report by removal of a narrowed portion of internal carotid with restoration by direct anastomosis to the common trunk. This would seem to be the ideal maneuver in such a situation; and it was unfortunate for our patient that she was not referred for treatment before her ischemic attacks culminated in cerebral infarction.

SUMMARY AND CONCLUSIONS

1. Three patients with carotid arterial disease are described in whom surgical treatment was not feasible: one who succumbed to fatal coma precipitated by ill-advised bilateral Diodrast angiography in the presence of widespread arteriosclerosis and one who recovered without any treatment; and another who, despite diffuse disease of the internal carotid that rendered arteriorrhaphy impossible, went on to develop satisfactory collateral supply following cervical sympathectomy (although we make no therapeutic claim for the operative procedure).

2. Analysis of a case of successful reconstruction of a thrombosed internal carotid artery, followed for over 4 years with comparable original and late postoperative angiograms, indicates that the neurologic effects of abrupt and poorly compensated occlusion may be ameliorated even by relatively late revascularization of the impoverished cerebral hemisphere.

3. Whenever the diagnosis of occlusion or insufficiency of the internal carotid artery has become established it is recommended that prompt investigation be directed toward the feasibility of resection of the involved segment, with or without thrombectomy or endarterectomy, for possible reconstruction by the method that seems to offer the best chance of success.

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* Dr. R. R. Linton states that arterial homografts are known to have functioned well for 3-4 years; autogenous vein grafts should function longer—he cites one such which continues to do well since its installation in 1950. Synthetic substances have also been utilized for carotid reconstruction (cf. papers by several authors, *Arch. Surg., Chicago*, 1958, *76:* 261–309).

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COMPLICATIONS IN THE SURGERY OF INTERNAL CAROTID ARTERY THROMBOSIS*

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ALLEVIATION of neurological symptoms following complete thrombosis of the internal carotid artery presents a difficult therapeutic problem. Surgical therapy has been approached in four ways: 1) excision of the thrombosed segment, 2) excision of the thrombosed segment with the interpolation of an arterial or venous graft, 3) end-to-end anastomosis of vessels, and 4) thrombo-endarterectomy. The rare occurrence of bleeding from the distal portion of the internal carotid artery has been documented in the experience of Webster *et al.*[7] in the exploration of 41 patients with demonstrated occlusion of the carotid arteries. Failure in attempted thrombectomy to effect retrograde bleeding from the internal carotid artery is illustrated in a case of bilateral thrombosis of the internal carotid artery and in a case of intracranial extension of thrombosis of the cervical internal carotid artery. The complications in the postoperative course of a patient who had retrograde bleeding from the internal carotid artery following thrombectomy suggested the advantage of performing thrombectomy through the common carotid artery. Retrograde flow of blood from the internal carotid artery was not a satisfactory index of complete removal of the thrombus in a case of this report. The hazards of anticoagulant therapy following successful thrombectomy are emphasized in the fatal hemorrhages found at necropsy in this patient.

CASE REPORTS

Case 1. A 58-year-old male had weakness of the left leg 1 year prior to admission. This gradually improved. A right facial weakness and hemiparesis began 2 days before admission to the hospital.

Examination revealed a right facial palsy, right hemiplegia, sustained right ankle clonus, and marked aphasia. Grade II hypertensive retinopathy was present.

Operations. Four months following admission left carotid angiography† and exploration of the cervical carotid artery revealed an organized thrombus in the common and internal carotid arteries. Thrombectomy was performed but there was no retrograde flow of blood from the internal carotid artery. Cervical sympathectomy was performed.

* Presented at the Medical and Surgical Conferences, Veterans Administration Hospital, Pittsburgh, March 19, 1957.

† Angiography in all cases was performed using 10–20 cc. per injection of 50 per cent sodium diacetrizoate (Hypaque Sodium).

Three weeks later right carotid angiography and exploration of the right carotid arteries in the neck revealed an organized thrombus in the common, internal and external carotid arteries which could not be completely removed. Vertebral angiogram revealed filling of the posterior cerebral artery on the right.

Course. Six months later the neurological findings had not changed from those on admission.

Case 2. A 41-year-old, right-handed male had sudden onset of loss of consciousness and left hemiplegia 1 week before admission.

Examination. He presented signs of mental confusion and had a complete left central facial palsy and left spastic hemiplegia. Spontaneous clonus of the arm and leg and Hoffmann and Babinski's signs were present on the left. Deep tendon reflexes were hyperactive, the left more than the right. Superficial pain sensation was intact. Touch sensation was decreased over the left extremities but preserved over the face. Motion and position sensations were lost in the left extremities.

Operations. Eighteen days after the onset of symptoms exposure of the internal carotid artery was carried out in the neck. Complete occlusion of the internal carotid artery was found at the bifurcation and a thrombus, 2 cm. in size, was removed. During removal of the clot the internal carotid artery was torn and this vessel was closed by sutures at the bifurcation. Anastomosis of external carotid artery to internal carotid artery was made above the site of this thrombosis. Free flow of blood from the proximal end of the external carotid artery and a good retrograde flow from the internal carotid artery were observed before the anastomosis was complete. Pathological diagnosis was arteriosclerosis of carotid artery.

Percutaneous right carotid angiography 3 weeks following operation revealed an occlusion of the common carotid artery. Exploration of the anastomosis disclosed a complete thrombosis of common, internal and external carotid arteries. Removal of thrombus from proximal and distal ends of the bifurcation did not result in a flow of blood.

Course. Ten months later he had increased spasticity in the left leg and arm. The right ulnar, radial and superficial temporal pulsations were absent. The popliteal, posterior tibial and dorsalis pedis pulses were not palpable bilaterally. Left superficial temporal and left carotid pulses were present.

Case 3. A 36-year-old, right-handed female presented with personality and mental changes beginning 1 week after parturition and 3 months before admission. During this period she had transient episodes of blindness. Three weeks following delivery she noticed a mass in the right breast which was treated with antibiotics and Meticorten, 20 mg. daily, for 15 days and the mass decreased about 50 per cent. Four days before admission the breast abscess was excised. Two days later she became emotionally agitated and this was followed by semicoma, right hemiplegia and aphasia.

Examination revealed a complete motor aphasia, left ptosis, right facial weakness, right hemiparesis, hypesthesia over the right half of the body and a probable right homonymous hemianopsia. Percutaneous carotid angiography revealed an occlusion of the left internal carotid artery.

1st Operation. Exploration of the left cervical internal carotid artery was performed 9 days after hemiplegia occurred. A thrombus, 5 cm. in length, was removed from the internal carotid artery. This partially organized clot began 2 cm. above the bifurcation of the common carotid artery. No retrograde blood flow was obtained

and repeated angiography on the operating table revealed a block in the artery at the base of the skull.

Pathological study of a biopsy of the sternocleidomastoid muscle revealed capillaritis of perimuscular connective tissue and acute lymphadenitis (jugular node) with bacterial masses in the node. This suggested hypersensitivity angiitis or infectious angiitis.

2nd Operation. Four days later the cervical carotid artery was re-explored and thrombus was found between the previous opening in the internal carotid artery and the common carotid artery. The internal carotid artery was torn during removal of the thrombus. Left frontal osteoplastic craniotomy under hypothermia revealed a thrombus in the supraclinoid portion of the internal carotid artery. Catheter and solution could not be passed retrograde in this vessel. There was no flow of blood from either middle or anterior cerebral vessels. The brain in the operative area was of normal appearance, but soft on palpation. Biopsy in the anterior portion of the superior temporal convolution revealed brisk arterial bleeding.

Course. Hemiplegia and aphasia persisted. Four days following operation her temperature rose to 106°F., which improved following tracheotomy. She became more comatose and expired 1 week following surgery.

Necropsy. There were organized thrombi in the left internal carotid, anterior and middle cerebral arteries. Microscopic study revealed that fibrinous thrombi extended distally into the smaller cerebral vessels. The brain on section revealed softening and liquefaction necrosis of the entire left cerebral hemisphere except for the occipital lobe. At the level of the mammillary bodies a fresh hemorrhage measured 5×2×1 mm. The cavernous sinus contained small early organizing thrombi and the adventitia was diffusely infiltrated with lymphocytes, plasma cells, and polymorphonuclear cells. The pituitary gland had several areas of recent infarction with minimal normal anterior pituitary lobe remaining intact. Multiple anemic infarcts were found in the cortical areas of the kidneys with thrombosis of the associated branches of the renal arteries. Focal thrombosis of the pulmonary arterioles, terminal bronchopneumonia, and recent infarcts of the papillary muscles of the heart were found on microscopic examination. Although the typical fibrinoid necrosis of the small arteries was not evident in this study, the pathological findings suggested a relationship to the so-called "intravascular coagulation syndrome."

Case 4. A 60-year-old male was admitted to the Medical Service with a history of shortness of breath, cough and precordial pain. There was a vague history of dizziness. While undergoing medical examination he had sudden onset of aphasia, right facial palsy, and right hemiparesis.

Examination. The patient was comatose. He had a right central facial palsy and right hemiparesis. No pulsation of the carotid sheath was detected beneath the left mandible. The left superficial temporal pulse and bilaterally the dorsalis pedis and posterior tibial pulses were faint to palpation.

Percutaneous angiography revealed a complete occlusion of the left internal carotid artery and partial occlusion of the external carotid artery (Fig. 1A). A normal vertebral arterial system was demonstrated.

Operation. Four hours after onset of symptoms the carotid sheath was exposed under local anesthesia. An unusual vascular pattern was present with the ascending pharyngeal artery originating between the external and internal carotid arteries and another vessel, arising behind the ascending pharyngeal artery at the bifurcation, coursed cephalad. A thrombus, 6 cm. in length, was removed with pituitary

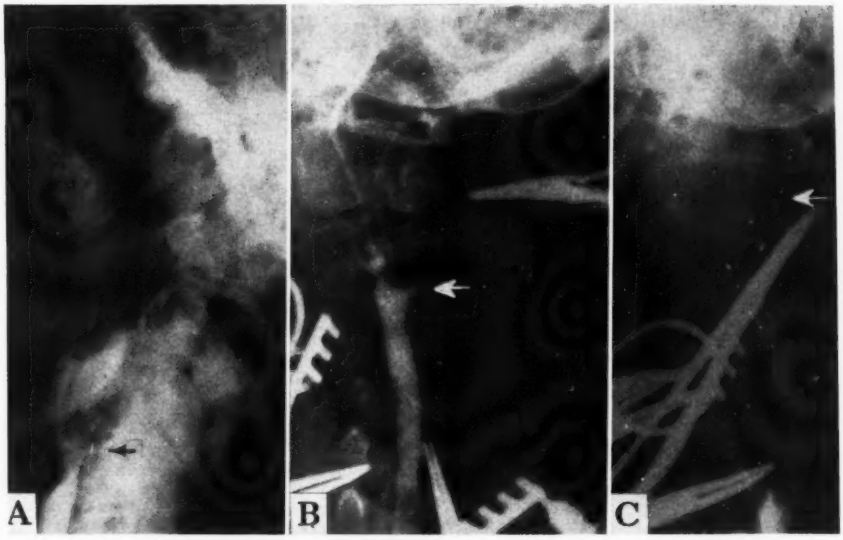

FIG. 1. *Case 4.* (A) Percutaneous angiogram demonstrating thrombus in common carotid artery and partial occlusion in external carotid artery. (B) Angiogram at operation demonstrates persisting thrombus in internal carotid artery after removal of thrombus 6 cm. in length. (C) Angiogram demonstrating persisting occlusion in higher levels in internal carotid artery after another attempt at removal of thrombus.

forceps and suction through a 1-cm. incision in the common carotid artery ½ cm. below the bifurcation. Fair retrograde bleeding occurred from external and internal carotid arteries. Angiogram made on the operating table revealed a persistent occlusion in the internal carotid artery at the region of the bifurcation of the common carotid artery (Fig. 1B). The artery was reopened and additional clot in the internal carotid artery was removed. Angiograms revealed occlusion of the internal carotid artery 4 cm. distal to the bifurcation (Fig. 1C). The artery was reopened and the suction was passed up the internal carotid artery and a partially organized thrombus, 5 cm. in length, was removed, followed by brisk bleeding from the vessel. Angiography 6 hours after the onset of symptoms revealed filling of the left middle and anterior cerebral arteries (Fig. 2). A small perforation was apparent on the posterior wall of the internal carotid artery 1 cm. from the bifurcation and bleeding was controlled with Gelfoam pack. The patient was given intravenous heparin after the first thrombus was removed.

Postoperative Course. A moderate hematoma developed in the left neck. Tracheotomy was performed to minimize respiratory embarrassment. A green string bean was aspirated from the lung at the time of tracheotomy. The patient showed progressive improvement in neurological findings. Within 24 hours he became alert and obeyed commands correctly. Forty-eight hours following surgery he had a complete return of motor function, including fine movements of the fingers. Speech was impaired because of marked swelling of the neck; however, 4 days postoperatively he was able to talk when the tracheotomy tube was occluded. He was maintained on Coumadin® (warfarin sodium) and followed by daily prothrombin times. Convales-

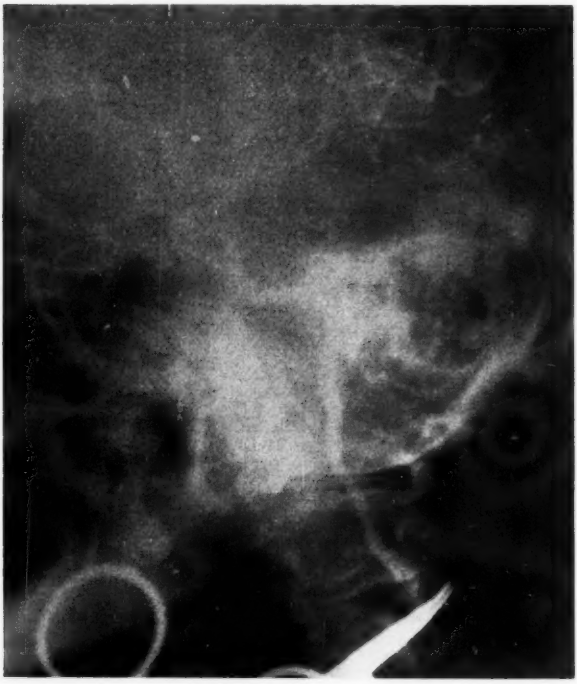

FIG. 2. *Case 4.* Angiogram demonstrates filling of anterior and middle cerebral arteries after removal with suction of the final thrombus, 5 cm. in length.

cence was uneventful but on the 10th day death occurred suddenly and unexpectedly. During the 8 hours prior to death blood-tinged mucus was aspirated from the tracheal tube; however, there was no hematuria. In the 3 days preceding death prothrombin time was 86, 76, and 77 seconds compared to the control of 13 seconds.

Necropsy. There was ecchymosis of the neck and chest incident to the previous hematoma. The left internal carotid artery was patent with a 3-mm. perforation in this vessel on its posterior wall 1 cm. above the bifurcation of the common carotid artery (Fig. 3). Grossly the brain appeared normal except for arteriosclerosis of the vessels of the circle of Willis. On section of the brain there was a recent hemorrhagic infarct in the left basal ganglia (Fig. 4). There were massive hemorrhagic areas in the lower lobes of the lungs and recent and old infarcts in the spleen. A healing septal infarct was found in the heart. Advanced pulmonary emphysema was present. The stomach contained fresh blood and bleeding could be identified from a chronic peptic ulcer on the lesser curvature of the stomach. There was minimal hemorrhage in the renal pelves. Death was attributed to the multiple hemorrhages as a result of a vascular diathesis incident to the anticoagulant medication.

DISCUSSION

Thrombosis of the carotid artery, as a manifestation of diffuse vascular disease, is illustrated in the cases cited. Thrombectomy does not alter the

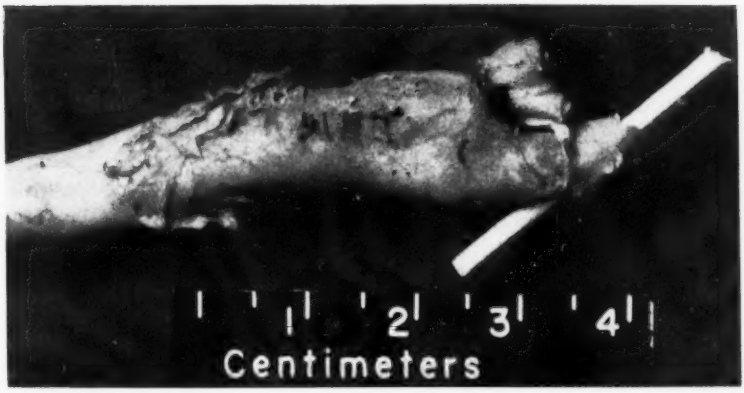

FIG. 3. *Case 4.* Photograph of region of bifurcation of left common carotid artery demonstrating the perforation in the posterior wall of the internal carotid artery and the sites of opening the common carotid artery for thrombectomy.

basic pathological process, so that one can anticipate further symptomatology from the vascular disease. It is difficult to predict whether appreciable improvement will occur spontaneously in the acute case with profound aphasia or paralysis following complete occlusion of the internal carotid artery, so that attempts at restoration of the circulation appear justified. Table 1 summarizes the individually reported cases of occlusion of the internal carotid artery treated by a variety of surgical methods. Although it

FIG. 4. *Case 4.* Photograph of recent hemorrhagic infarct in the left basal ganglia.

TABLE 1

Summary of reported cases of carotid artery thrombosis treated surgically

Source Date	Etiology	Percentage Block	Time Before Surgery	Procedure	Result
Strully *et al.*[6] 1953	A-S	100%	3 days	T.E.	No change
Eastcott *et al.*[3] 1954	A-S	Partial	Intermittent symptoms	Anastomosis internal to common carotid artery	Free of symptoms
Denman *et al.*[2] 1955	A-S	100% bilateral	3 months	Arterial graft	Improved
Lin *et al.*[5] 1956	A-S	Partial	Intermittent symptoms 7 years	Vein graft	Improved
Cooley *et al.*[1] 1956	A-S	Partial	4 months	T.E.	Improved
Edwards and Rob[4] 1956	A-S	Partial	2 months	Anastomosis internal to common carotid artery	Improved
Arnstein[8] 1957	A-S	100%	2 weeks	T.E.	No change
	A-S	100%	10 days	T.E.	No change
	A-S	50%	? 9 months	T.E.	80% improved
	? T.A.O.	75%	1 year	T.E.	Less than 50% improved
	A-S	100%	3–4 days	Suction	100% improved
	A-S ? T.A.O.	100%	12 hours	T.E.	80% improved
	Trauma	75%	? 1 month	T.E.	Less than 50% improved
	Unknown	50%	3 days	T.E.	50% improved (not immediate)
	S.B.E.	100%	8 hours	Suction & T.E.	90% improved
Lemmen *et al.* 1958	A-S	100% bilateral	4 months	T.	No change
	A-S	100%	18 days	Anastomosis external to internal carotid artery	No change
	? i.c.s.	100%	9 days	T—cervical & intracranial	No change—7 days death
	A.S.	100%	6 hours	T & suction	Improved 10 days —death

A-S = arteriosclerosis, T.A.O. = thromboangiitis obliterans, S.B.E. = subacute bacterial endocarditis, T.E. = thrombo-endarterectomy, T. = thrombectomy, i.c.s. = intravascular coagulation syndrome

is difficult to evaluate from these data whether surgical therapy significantly improves these patients, it seems likely that younger individuals with limited vascular disease would derive the greatest benefits. Success of surgical therapy in alleviating neurological symptoms secondary to thrombosis is dependent in part on prompt diagnosis and removal of the clot. Reported experiences do not define the maximum allowable time-interval between development of complete occlusion of the carotid artery and successful thrombectomy in effecting neurological improvement.

In the 2 cases in which thrombus was excised through the internal carotid artery the operation was complicated by a tearing of this vessel. In order to correct this difficulty in Case 2 an end-to-end anastomosis between internal and external carotid artery was carried out. The complication of a retrograde thrombosis from this anastomosis was illustrated in the resultant surgically induced "pulseless" disease on the involved side. This tearing of the internal carotid artery could have been avoided by the use of an incision into the bulb of the common carotid artery as illustrated in Case 4. Since this latter vessel has a larger diameter it permits suture with less embarrassment of the lumen. Ligation of the external carotid artery was performed in this latter case to transmit the full head of arterial pressure into the internal carotid artery. Suture of the opening in the internal carotid artery was not deemed advisable because of the arteriosclerotic process at the site of penetration. Although the method employed affords an ideal situation for the development of a false aneurysm, the critical state of the patient at operation precluded attempts at graft or anastomosis.

The role of anticoagulant therapy as an adjunct to surgical therapy is not clearly defined at the present time in cases of thrombosis of the carotid artery. Anticoagulant therapy was considered the factor responsible for the recent multiple hemorrhages noted in the necropsy study of Case 4 and emphasizes that it is not without hazard. Difficulty in regulation of prothrombin time was encountered in this patient. While this medication may have been a factor in the patency of the internal carotid artery, a shorter period of anticoagulant therapy might have been equally as effective.

Retrograde flow from the internal carotid artery was not a satisfactory index of complete removal of the thrombus. A ball-valve type of mechanism by the persisting thrombus appeared to be a logical explanation for the observed retrograde flow of blood from the internal carotid artery while complete block was demonstrated by angiography. A retrograde flow of blood occurred in Case 2 which was similar to that seen in Case 4 before the complete removal of the thrombus. In retrospect a persistent thrombus higher in the internal carotid artery in Case 2 could have contributed to the extensive thrombosis found at the second operation. Angiography should follow all cases in which anastomosis or graft is performed to restore circulation of the internal carotid artery. Thrombectomy is best carried out by opening the common carotid artery below the anastomotic site. Persisting thrombus in the lower portions of the internal carotid artery may be removed with a metal suction tip. Polyethylene tubing can be passed further cephalad into the vessel and a trial of suction and irrigation with saline through this tubing may on occasion successfully relieve higher occlusions in the artery. The operative procedure is facilitated by employment of serrefine clamps. Preparation should be made to counteract promptly the variable loss of blood associated with this procedure. Postoperatively one must be alert to possible rupture of the diseased vessels or the anastomosis.

Intracranial approach to the thrombus in the supraclinoid portion of the

internal carotid artery was complicated by the development of vasospasm from manipulation of the vessels despite hypothermia. We were not able to remove the thrombus from the cavernous portion of the internal carotid artery nor could the obstruction be relieved in the collateral circulation between anterior and middle cerebral arteries. Hypothermia was employed in order to reduce the chance of permanent cerebral damage had these vessels been patent and had it become necessary to temporarily occlude these latter vessels. This would be particularly important should injection with contrast media of the contralateral carotid system reveal patency of the anterior and middle cerebral arteries through the anterior communicating vessel, as illustrated in the angiograms of Strully *et al.*[6]

SUMMARY

Thrombosis of the carotid artery, as a manifestation of diffuse vascular disease, is illustrated by the cases of this report. Limitations in surgical therapy were demonstrated in a case of bilateral thrombosis of the internal carotid artery and in a case of intracranial extension of thrombosis of the cervical internal carotid artery. The advantages of thrombectomy through the common carotid artery were indicated. Retrograde flow of blood from the internal carotid artery was not an index of complete removal of the thrombus and the importance of concurrent angiography is emphasized. The hazards of anticoagulant therapy following successful thrombectomy are illustrated in the fatal hemorrhages found at necropsy in a case of this report.

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EXPERIENCE WITH SURGICAL TREATMENT IN TWENTY CASES OF PITUITARY ADENOMAS*

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RECENTLY, occasion arose to review the cases of hypophysial adenoma treated surgically at the Ochsner Clinic during the 12-year period from its founding on Jan. 1, 1942 through Dec. 31, 1953. All operations were performed by me in accordance with an arrangement whereby pituitary operations done elsewhere at the teaching hospitals affiliated with the Tulane Medical School were performed by my associates. Although this 12-year experience with private patients having pituitary tumors might lead the reader to expect an analysis of 50 or more cases, there were only 20 patients, and one of these had been operated on previously by another surgeon. In addition to this group of patients treated surgically, 10 patients with pituitary adenomas received deep roentgen-ray therapy only.

There are two chief reasons for this relatively small experience with pituitary adenomas. First, patients with the classical pituitary adenoma syndrome of bitemporal hemianopsia and an enlarged sella turcica are being referred directly to specialists in roentgenotherapy, who are now available in all but the smaller communities. Secondly, there are now approximately 850 practicing neurosurgeons in the United States, among whom the limited number of patients with pituitary adenomas is being distributed. No longer can an American neurosurgeon expect to accumulate a large series of cases of pituitary adenomas or any type of neurosurgical case other than ruptured intervertebral disk and head injury. This means that in the coming decades most neurosurgeons will have relatively limited experience with brain tumors, spinal cord tumors, trigeminal neuralgia, and the other diseases that once monopolized the neurosurgeon's time. As a consequence, most neurosurgeons will devote an ever increasing portion of their time to complex neurological problems, such as intractable pain, headache, epilepsy and abnormal involuntary movements, which may or may not require surgical treatment.

TWO SURGICAL DIVISIONS OF PITUITARY TUMORS

Most writers have divided pituitary adenomas into two groups: those confined to the immediate region of the sella turcica and those with extension of a large portion into the brain or nasopharynx. There is good reason

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for this division because the surgical problems presented by the two types are unrelated and the mortality rate is much higher in the latter group. The mortality rates for the large tumors were 35 per cent in Olivecrona's series,[1] 33 per cent in Jefferson's series,[6] and 34 per cent in Horrax's series.[5]

ADENOMAS WITH CEREBRAL EXTENSIONS

Only 2 of my 20 patients had pituitary adenomas with large portions extending into the brain. The great size and formidable location of the lesions were demonstrated pneumoencephalographically and confirmed surgically as well as at necropsy. Both of these patients (Mrs. M.A.S., aged 27 years and Mr. C.C., aged 59 years) were stuporous prior to operation. In retrospect, these operations, performed in 1943 and 1946, were not well planned. The goal was to remove at one operation as much tumor as possible and to preserve the small amount of remaining vision. Probably it would have been better to plan a two-stage operation with removal of part of one frontal lobe and part of the tumor first. If this had resulted in restoration of consciousness and normal intracranial pressure, another operation could have been performed to remove still more of the tumor and decompress the optic apparatus.

Jefferson,[6] White and Warren[9] and others have written in an illuminating manner on the surgical management of these large tumors. Jefferson pointed out that operation on these patients is a course of doubtful wisdom and not often beneficial to the patient.

An example of nonintervention is the case of R.S., whom I examined in 1955. She had been successfully treated for a pituitary adenoma elsewhere many years before. The original operation had corrected the bitemporal hemaniopsia and had enabled her to continue as manager of a large business office. Shortly after her retirement at the age of 65 years her sight began to fail, and when she consulted me, she was nearly blind. In spite of absence of manifestations of increased intracranial pressure, pneumoencephalograms showed extension of a large portion of the tumor into the right frontal lobe. Her insistent request for surgical treatment was not granted because she was healthy and happy and she had no living relatives to care for her if invalidism developed. This decision has thus far proved to be wise. When last seen on Oct. 20, 1956, she was still well and completely adjusted to total blindness.

ADENOMA CONFINED TO REGION OF SELLA TURCICA

The remaining 18 patients of the 20 in this series had relatively small adenomas confined to the immediate region of the sella turcica.

One of them (W.P.K.), a man aged 41 years, was operated on in 1941 by the late Walter Dandy, a left-sided approach having been used because vision was poorer on that side. Transient aphasia had followed operation but vision had promptly returned to normal.

When the patient consulted me 8 years later, he was again unable to read with the left eye and there was no vision in the temporal field of the other eye. At operation in 1949 via the old left-sided flap, an "empty" sella turcica was found. How-

ever, there was obviously extrasellar adenomatous tissue because the left carotid artery was displaced upward and medially, and this had, in turn, caused such acute angulation of the left optic nerve at its foramen that it was necessary to unroof the nerve into the orbit. The other optic nerve was not seen. Again, transient postoperative aphasia developed. Roentgen radiation was given for the first time.

When the patient was last examined 5 months after operation, the vision in the left eye was improved but bitemporal hemianopsia persisted. In August 1956 the patient wrote that vision had not deteriorated during the past 7 years and that he has "no trouble reading the newspapers and watching the girls." He is possibly correct in attributing the satisfactory results to the postoperative roentgenotherapy rather than to the operation.

Of the remaining 17 patients, 15 were operated on for failing vision and 2 for incapacitating headache. There were no deaths. Obviously, this has no statistical significance in view of the small number of cases. The operative mortality rate has always been low with this type of lesion, as indicated by a rate of 3.9 per cent in Horrax's[5] series, 6.4 per cent in Olivecrona's[1] series and 2 per cent in Jefferson's[6] group. All but one of my 18 patients with small adenomas are still alive.

The patient (A.S.), a tailor 53 years old, had bitemporal hemianopsia that did not improve with radiation; actually, roentgenotherapy was discontinued because of decrease in visual acuity and enlarging bitemporal scotomata. At operation the adenoma consisted of a capsule containing chocolate-colored fluid. The capsule was completely dissected from the optic nerves and chiasm with resulting return of the visual fields and visual acuity to normal. The patient continued to work as a tailor until his death 17 months later from myocardial infarction.

Indication for Operation. It is my opinion that pituitary adenomas should be treated by radiation unless vision is already seriously threatened bilaterally. Of the 18 patients with adenomas confined to the immediate region of the sella turcica 6 were operated on because irradiation had failed to improve vision or relieve headache. The remaining 12 were operated on without a trial of irradiation because of bitemporal hemianopsia. Nine of the 12 also had one partially blind eye.

Vision Made Worse by Operation. The only patient (L.A.W.) whose vision was made worse by operation was a man 22 years old who had bitemporal hemianopsia of 6 months' duration and headache.

The adenoma, exposed through a right-sided craniotomy in 1946, proved to be solid and vascular. A retrochiasmal nodule was pulled down and under the optic chiasm in the manner described by Cushing.[3] Complete blindness of the contralateral eye and ipsilateral 3rd nerve palsy resulted. The latter subsided in 6 months. Radiation therapy is still being kept in reserve.

When the patient was last seen in September 1956, he was working and in good health. He cautiously drives a car in a rural area with nothing more than central and nasal vision in one eye. He reads slowly, never for pleasure. Inasmuch as the patient and his relatives believe that the result of the operation was miraculously successful, only the surgeon is disappointed.

Cases of Recurring Visual Failure. In 3 patients visual failure recurred at intervals of 8, 6, and 3 years after operation.

One of them (W.O.), a man aged 33 years, was operated on in 1946 for a cystic adenoma that was causing bitemporal hemianopsia, without stigmata of hypopituitarism. When seen 5 years later, he could read with either eye but he still had no temporal vision in the right eye. Eight years after operation, the patient returned with blindness of the left eye and poor vision on the right. His vision had been failing for 6 months but he had been afraid to undergo another operation.

On Dec. 29, 1954 the chiasmal region was approached from the opposite (left) side. A large arachnoidal cyst containing 10 cc. of bright yellow fluid was found to be the principal cause of the failing vision. After the cyst was removed, the capsule of the adenoma was reopened and found to be empty. Parts of the capsule were dissected away, but apparently too much traction was exerted because 3rd nerve paralysis on the contralateral side appeared after operation.

Although the patient went home on the 7th postoperative day, he returned 11 days later with meningitis, from which he completely recovered. The last correspondence with him in September 1956 indicated that he was well and driving a tractor at a paper plant. He still had bitemporal hemianopsia but had no trouble in reading. The oculomotor nerve palsy disappeared. He denied having symptoms of hypopituitarism.

The second patient (W. S.), a man aged 44 years, was operated on in 1946 for a solid adenoma of the pituitary gland that had caused blindness on the left and loss of vision in the temporal field on the right during a period of 3 years. At operation the tumor not only bulged upward between the optic nerves but also protruded between the right optic nerve and right carotid artery. It was not adherent to the optic nerves and chiasm, and reasonably good decompression of these structures was accomplished. Roentgenotherapy was given a few weeks after operation.

In spite of periodic ophthalmologic examinations, the patient had to be led into my office 6 years later. His eyesight had been failing for 5 months and vision had disappeared abruptly from the useful eye (right) the day before. At operation on the next day the tumor was not much bigger than it had been originally but contained 10 cc. of chocolate-colored fluid. The right optic nerve was completely decompressed and the left optic nerve, which had long been useless, was severed; this permitted the right optic nerve and optic tract to migrate a few millimeters away from the thick capsule of the adenoma. Vision in the right eye gradually returned, and the patient went back to work in 60 days.

When last examined in October 1956, he had 20/20 vision in the right eye but a constricted temporal field. He was well, had no symptoms of hypopituitarism and was working as a salesman in a clothing store.

The final patient (E.L.S.), a woman aged 62 years, to have recurrence of visual failure after operation was operated on in 1951 because of bitemporal hemianopsia caused by a solid adenoma of the pituitary gland. A pneumoencephalogram was made preoperatively because of mild mental confusion, which was later attributed to barbiturates. The capsule of the adenoma was tight and some of the soft contents escaped when it was incised. Conservative and reasonably satisfactory decompression of the optic nerves was accomplished, and the patient left the hospital on the 7th postoperative day. The bitemporal hemianopsia disappeared and her

only residual complaint was complete anosmia. Roentgenotherapy was completed 6 weeks after operation.

Nearly 3 years later she was again having trouble reading, and a second course of irradiation was given. Correspondence with the patient's brother and physician in October 1956 disclosed that she had just completed her third and final course of roentgenotherapy. She is still able to read but has lost interest in reading. Her memory is said to be poor; she is slightly confused at times, and does not eat well. She may be becoming senile at the age of 68 years.

Operation for Incapacitating Headache. Two patients had no visual disturbance but were operated on because of incapacitating headaches and enlarged sella turcicas. One had acromegaly. Roentgenotherapy failed to relieve the headache in both cases.

The acromegalic patient (C.A.), a man aged 28 years, had not worked for 2 years because of severe bitemporal and frontal headache. At operation the capsule of the adenoma was found to be slack, and removal of its contents had no effect on the headache. However, the patient began working part time 1 year after operation, is now working full time as a policeman, and has noted 50 per cent improvement in the headache. Hypogonadism has persisted.

The other patient (R.B.), a man aged 28 years, had a pneumoencephalogram (normal) and bilateral arteriograms (normal) because unexplained somnolence developed 1 year after the ineffective irradiation. At operation the diaphragm of the sella was hidden by large veins and was not opened. Paradoxically, the headache was relieved, and some of the less annoying symptoms are now controlled by testosterone. When last seen 6 years later in September 1956, the patient was still well, working full time, and complaining only of decreased sexual vigor.

ENDOCRINOLOGIC FACTORS

Chromophobic adenomas of the pituitary gland that have caused expansion of the sella turcica and have recently compressed the optic nerves and chiasm rarely produce much clinical or laboratory evidence of hypofunction of the endocrine glands. Decrease in libido and potentia in men and amenorrhea in women are the commonest endocrinologic symptoms. Low basal metabolic rate, obesity, loss of sexual hair, hypotension and anemia are sometimes present in surgical patients but overt failure of the adrenal glands rarely has taken place at the time that vision begins to fail. In brief, although the patient with a chromophobic adenoma may have some subjective or objective stigmata of hypopituitarism, complete investigation by the endocrinologist rarely discloses laboratory evidence of serious pituitary insufficiency. Nevertheless, every candidate for surgical or roentgen-ray therapy of a hypophysial adenoma should be studied by an endocrinologist and observed at regular intervals thereafter.

In view of this relatively normal function of the pituitary gland despite physical compression and distortion by a chromophobic adenoma, it is not surprising that most patients subjected to surgical removal or irradiation of the adenoma have always done well without support from such agents as

corticotrophin or cortisone. However, during the past 6 years most surgeons have been giving ACTH during the period of surgical stress as a routine precaution. It is not possible to know whether this is of real value in the majority of cases, but Raaf and coworkers[8] obtained the impression that such medication decreases postoperative edema of the face, and presumably of the brain, and helps control the postoperative fever. It was the impression of Horrax and associates[5] that ACTH decreases the possibility of fatal hyperthermia.

Only 2 patients in this series (R.E.P. and J.T.) had serious postoperative disturbances of pituitary function.

The former, a man aged 58 years, had blindness in one eye, a temporal defect in the other and moderately severe headaches. At operation in 1950 a solid adenoma with retrochiasmal extension was removed. The course was stormy and he did not leave the hospital until the 20th postoperative day. He was drowsy, thirsty and incontinent. Probably, these symptoms were caused primarily by injury of the hypothalamus.

Nothing was known about the patient until 6 years later when a diagnosis of severe hypopituitarism was made elsewhere. In October 1956 the patient's son reported that the patient was working, happy and reasonably well except when his supply of thyroid extract and cortisone became exhausted. His vision was about the same as before operation and he was able to read.

The other patient (J.T.), a man aged 47 years, had removal of a solid adenoma including a retrochiasmal extension on Feb. 4, 1953. This lesion had caused blindness of one eye, temporal blindness of the other and mild headaches. The nerve to the already blind right eye was sacrificed in order to accomplish adequate decompression of the chiasm. The postoperative course was stormy and the patient was drowsy and disoriented for several days. The blood pressure was low, and thirst and urinary output increased. The temperature did not rise above 104°F. When the patient was able to leave the hospital on the 21st postoperative day, he was still lethargic and disinterested. It was presumed that these symptoms were caused by injury of the hypothalamic region.

The patient is now receiving a maintenance dose of Hydrocortone Acetate® and Depo-Testosterone®. In October 1956 he was still reasonably well, working long hours and free of complaints. Vision in one eye is normal except for a field cut in the lower temporal quadrant.

PERTINENT FEATURES IN REMAINING CASES

Of the 20 cases of pituitary adenomas treated surgically, 12 have been described briefly or at some length. The remaining 8 cases* are of no special interest and can be summarized as follows. Three were operated on in 1948, 1 in 1949, 2 in 1950, 1 in 1952, and 1 in 1953. Two had acromegaly, all had bitemporal hemianopsia, 5 had one blind or nearly blind eye, all obtained improvement in vision which was still being maintained in August 1956 or later, and all are active, working and reasonably well. The follow-up periods

* R.P., female, 69 years; I.B., female, 39 years; J.H., female, 46 years; A.B. female, 52 years; M.S., male, 37 years; C.P., female, 26 years; M.H., female, 46 years, and W.M., female, 69 years.

for these 8 patients ranged from nearly 3 to 8 years. Only the 2 acromegalic patients have overt evidence of pituitary dysfunction.

ANGIOGRAPHY AND PNEUMOGRAPHY

In recent years much has been learned about the appearance of pituitary adenomas in pneumoencephalograms and angiograms as explained by List,[7] Gurdjian *et al.*,[4] Bakay and White[2] and others. Apparently, these diagnostic procedures are being performed frequently in many hospitals. On the other hand, Horrax *et al.*[5] have been reluctant to carry out such investigations in patients with definite clinical evidence of small adenomas impinging on the visual apparatus. However, all agree that pneumoencephalography or cerebral angiography is indicated if the symptoms are atypical and compatible with such diagnoses as craniopharyngioma, large aneurysm, meningioma of the tuberculum sellae or extrasellar extension of a pituitary adenoma. In order to rule out lesions simulating small pituitary adenomas, pneumoencephalograms were made on 6 of the 20 patients in this series, including the 2 who had huge extensions of the adenoma into the frontal lobe. Only 1 patient (R.B.) had bilateral arteriograms.

TECHNICAL CONSIDERATIONS

In this series the incisions in the scalp were either of the coronal type or unilateral and just behind the hairline. The bone flaps were small, fashioned with four burr holes, and medially placed. A left-sided flap was used on four occasions because vision was nearly absent on that side. In one instance the 1.5-inch circular trephine was satisfactorily used but an opening of this size could be a handicap if complications arose. The dura mater was opened near the edge of the bone flap instead of at the sphenoid wing. No attempt was made to close the dura mater tightly. Although preoperative spinal drainage was carried out in all cases, in 2 instances partial right frontal lobectomy was necessary to obtain adequate exposure. The olfactory nerve was frequently avulsed when the frontal lobe was retracted. Indeed, on three occasions both olfactory nerves were avulsed.

On two occasions the optic nerve to a nearly blind eye was deliberately sacrificed in order to relieve tension on the chiasm and functioning optic nerve. On three occasions a retrochiasmal nodule was delivered by pulling it down and under the chiasm. However, it is possible that a retrochiasmal extension of the tumor was overlooked on one or more occasions. The anterior cerebral artery has not been severed between silver clips.

The capsule of the adenoma was usually coagulated and opened between the optic nerves but in 2 cases the tumor also presented itself between the optic nerve and carotid artery and was also opened there. One adenoma was opened posterior to the anterior cerebral artery as well as between the optic nerves. On several occasions decompression of the optic chiasm and nerves has been inadequate because of the vascularity of the tumor or because of the thickness of the capsule. Indeed, the contralateral optic nerve was not always

seen. Possibly I have been too conservative in the attack on the adenoma but the danger of damage to an optic or third nerve has been the restraining factor. No patient was returned to the operating room because of suspected intracranial hematoma but this was seriously considered in one instance (J.T.).

FOLLOW-UP STUDIES

The 2 patients having large extensions of pituitary adenomas into the cerebral hemisphere died in the hospital after operation. Another patient died of myocardial infarction 17 months after a successful operation. On Aug. 1, 1956, the remaining 17 patients were alive; thus, the survival period for these ranged from approximately 3 to 10 years. All 17 had vision and all but 1 were working. This woman (E.L.S.), aged 68 years, has become senile.

SUMMARY

Twenty consecutive patients with hypophysial adenomas were treated surgically during the 12-year period ending Dec. 31, 1953. Ten others received deep roentgen-ray therapy only. The only 2 patients whose adenomas invaded the brain were stuporous before operation and died in the hospital. One patient died 17 months after operation of myocardial infarction. In the other 17 patients vision was being maintained on or after Aug. 1, 1956, a follow-up period of approximately 3 to 10 years. All patients are working except a woman, 68 years old, who has become senile. Pituitary adenomas should be treated by roentgen-ray therapy unless vision is already seriously threatened bilaterally.

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CASE REPORTS

OSTEOMYELITIS OF CERVICAL VERTEBRAE (AND QUADRIPARESIS) SECONDARY TO URINARY TRACT INFECTION

CASE REPORT AND REVIEW OF LITERATURE

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Metastatic osteomyelitis of the vertebral column secondary to infection of the urinary tract has been recorded in the literature since 1925 in a total of 19 cases. Bacteriologic proof of the same organism in both the urine and the bony lesion has been demonstrated in only 5 cases. The present report describes an additional case with bacteriologic proof.

The first mention of the relation between vertebral osteomyelitis and infection of the urinary tract apparently was made by Goldschmidt[8] in 1925 (reported by De Feo[5]). He felt, however, that the infection of the bone preceded the urinary infection. A causal relationship between urinary infection and spinal osteomyelitis was first given serious consideration by Carson[3] in 1931, who recorded 4 cases, in 2 of which he obtained cultures of *Staphylococcus aureus* from both the urine and osseous infections. The cases recorded in the literature since Carson's report are listed in Table 1.

TABLE 1

Cases of vertebral osteomyelitis and urinary infection

Name of Authors	Year	Number	With Bacteriologic Proof
Carson[3]	1931	4	2
Kretschmer & Ockuly[10]	1935	1	
Kulowski[11]	1936	1	
Kusunoki[12]	1938	1	1
Schein[14]	1940	1	
Deming & Zaff[6]	1943	3	
Donahue[7]	1949	2	
Hurwitz & Albertson[9]	1950	1	1
Adlerman & Duff[1]	1952	1	
De Feo[5]	1954	2	
Mussey[13]	1954	2	1
Ablin & Erickson	1958	1	1
Total		20	6

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CASE REPORT

#295011. H.W., a 61-year-old white male, was re-admitted to University Hospitals on May 5, 1954. Previously a transurethral resection for benign prostatic hypertrophy had been performed on March 31, 1954. The patient's postoperative course had been relatively uneventful except in retrospect. On the day of surgery and the 1st and 2nd postoperative days he had had a fever up to 101°F., and a white blood count of 10,400/ml. with 88 per cent polymorphonuclear cells. On the 3rd day he had a chill with a temperature of 104°F. He had been on 300,000 units of procaine penicillin (Duracillin) and 0.5 gm. streptomycin twice daily. This was stopped on the 3rd day and he was started on 250 mg. oxytetracycline hydrochloride (Terramycin) and 0.5 gm. sulfisoxazole (Gantrisin) every 6 hours. On the 4th day the temperature came down to as low as 99.8°F. The 5th day was uneventful and without fever. However, on the 6th postoperative day for the first time the patient complained of pain in the back of his neck. He was unable to sleep that night. The pain in the neck persisted for 3 more days. On the 10th postoperative day he was apparently more comfortable and was discharged. It is interesting to note that the pain was so severe the day before discharge that the nurses recorded that he was pacing the floor because of the discomfort in his neck. He had been completely afebrile for the last 5 days of his hospitalization.

The remainder of the past history, systemic review and physical examination were noncontributory. Microscopic study of the prostatic tissue removed during surgery revealed adenomatous hyperplasia with patchy prostatitis. Acini in some fields had collars of inflammatory cells. Roentgenograms of the cervical spine on April 8, 1954 were reported to show quite marked osteoarthritis of the cervical spine with slight scoliosis. He was discharged on April 10, 1954.

On about April 25, 1954 he noted hematuria and was admitted to a local hospital. He was re-hospitalized there on May 3, 1954 because of difficulty in walking. From May 3 through May 5, 1954 he had progressive symptoms of motor and sensory loss in his upper and lower extremities. The patient had noted progressive increase of pain in his neck and in the right shoulder from April 6 through May 5, 1954.

Examination on readmission to the University Hospitals, May 5, 1954. There were no significant changes in general physical findings. His temperature was 101.2°F. Neurological examination revealed an asymmetric quadriparesis with a sensory deficit for light touch and pinpoint at about the C8 dermatome and extending across the chest just below the clavicles bilaterally. The superficial reflexes were lost; deep tendon reflexes were present symmetrically and within normal limits. There was pain in moving the right shoulder girdle and marked tenderness over the lower cervical spines. There was a level of anhidrosis quite evident below the clavicles. No anisocoria was noted.

Roentgenograms of the spine showed erosion of the 6th cervical vertebral body. Lumbar puncture and Pantopaque myelography revealed a complete block at the C6 level (Fig. 1). Routine laboratory studies revealed a marked pyuria. He had a hemoglobin of 11.6 gm. and 11,300 white blood cells/ml. with 67 per cent polymorphonuclear cells. Cultures of the urine now yielded *Streptococcus fecalis* and *Pseudomonas aeruginosa.*

Course. The patient showed further evidence of progressive paresis. On May 6, 1954 cervical traction with the use of Crutchfield tongs was instituted.

1st Operation. On May 10, 1954 a decompressive laminectomy of C3 through C7 was performed (Fig. 2). An epidural granulomatous lesion producing complete block at C5 with dorsal displacement of the dura mater and its contents was disclosed. After this the dura mater was noted to pulsate well.

2nd Operation. The next day, on May 11, 1954, because of the opinion that the myelogram suggested an intradural lesion, the wound was opened and the dura mater was incised. The cord appeared to be displaced backwards in the mid-cervical region. No intradural, extramedullary mass was observed either dorsally or ventrally after thorough exploration. Aspiration with a No. 26 hypodermic needle in the dorsal midline of the cord revealed no intramedullary abnormality. The dentate ligaments on both sides were sectioned to facilitate decompression.

Studies of Surgical Specimens. Cultures and sensitivity studies of tissue removed at the time of the laminectomy revealed the presence of *Pseudomonas aeruginosa* sensitive to polymyxin. Biopsies of the vertebrae taken at the time of the laminectomy showed a destructive inflammatory process and chronic osteomyelitis.

Postoperative Course. After a short course of procaine penicillin and streptomycin, the patient received 3.3 gm. of polymyxin[B] over a period of 22 days until June 11, 1954, at a rate of about 150 mg. per day (2–3 mg./kg.). Emergency tracheotomy was necessary on May 21, 1954.

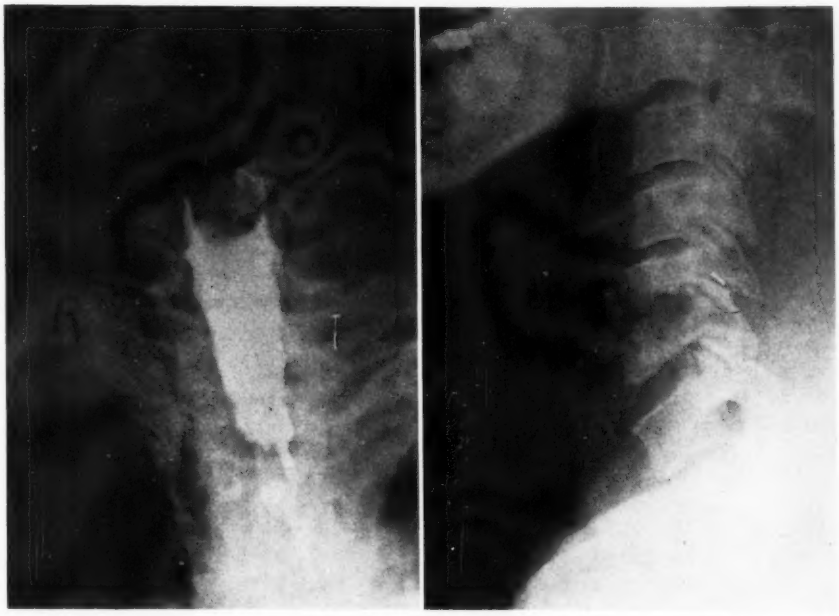

FIG. 1 (*left*). Preoperative oil myelogram showing complete block in the low cervical region.
FIG. 2 (*right*). Postoperative film of cervical spine showing defect in bodies of C5 and C6 as well as operative removal of spines of C3 through C7.

Because of progression of the osteomyelitis upward to C3 and the radiological finding of a retropharyngeal prevertebral soft-tissue swelling, exploration of the retropharyngeal space and the upper and mid-cervical vertebrae was performed on July 9, 1954. No abscess was encountered but biopsy of these abnormal vertebrae was done and culture yielded the pyocyaneus organism again. He received another short course of polymyxin[B] and his temperature gradually came down to normal by about the 15th postoperative day. The operative wounds all healed well. Periodic roentgenograms of the cervical spine showed cessation of the progressive osteomyelitis, regression of the prevertebral swelling and, finally, recalcification of the destroyed vertebrae with bony proliferation. The soft-tissue space between the pharynx and cervical spine decreased and was thought to be normal on Aug. 10, 1954.

Following this last operative procedure, the patient showed signs of some sensory and motor improvement. There was a residual dissociated loss of pinpoint and light-touch sensation. The almost complete paraplegia improved to a point where he could flex the left knee and wiggle the toes. He was able to lift the left leg off the Stryker frame against gravity while

lying on his abdomen. The knee and ankle jerks were present bilaterally; the biceps reflexes improved to almost normal while the triceps remained hypoactive. Crutchfield traction was removed on Aug. 14, 1954, 3 months after placement. He was discharged on Oct. 29, 1954. When last heard from on April 1, 1955 there had been no significant change except slight further motor improvement.

DISCUSSION

It was felt that this patient's illness represented a metastatic, septic osteomyelitis of the cervical spine, probably occurring at the time of or shortly after his transurethral resection through one of the open, enlarged prostatic sinuses, with septic urinary and prostatic contents traveling by way of the vertebral venous system.[2] Undoubtedly the first symptom of this was the pain in his neck, 6 days after his transurethral resection, with progression described above. It should be noted that the same organism (*Pseudomonas aeruginosa*) was cultured repeatedly from the pathological lesions in the cervical spine and from the urine.

Creevy and Feeny,[4] in an excellent study of 94 patients immediately following transurethral resection of the prostate, demonstrated positive blood cultures in 45.6 per cent (43 patients) who had not received antibiotics prior to the surgical opening of the large prostatic venous sinuses in the presence of infected urine.

Batson[2] in a classic paper demonstrated the existence of a pathway for the spread of possible infection or neoplastic cells from the dorsal vein of the penis and pelvic veins via the bone-protected vertebral venous system of the spinal column, along the entire spinal column up to the skull, and completely by-passing the lungs and inferior vena cava. With increased intra-abdominal or intrathoracic pressure he proved that return of blood can occur via the vertebral system rather than the inferior vena cava. (It is to be noted that of the 20 patients mentioned, at least one-half of them had known abdominal distension, hiccoughs, coughing, straining, or wore abdominal binders.)

The work of these investigators, when coupled, might account for the spread of infection of the urinary tract following instrumentation via the vertebral venous system to the vertebral column. It would also explain the occurrence of spinal osteomyelitis following urinary infection in decreasing order of frequency in lumbar, dorsal, cervical, and sacral areas. Further this review suggests that the frequency of relationship between vertebral osteomyelitis and antecedent urinary infection following instrumentation may not have been sufficiently recognized in the past; one may anticipate finding a greater number of such cases with the awareness of such a probability. In many cases it is not practical to demonstrate the organism in the spinal lesion and this consideration, too, leads one to suspect its occurrence more frequently than demonstrated so far.

SUMMARY

1. A review of the literature of metastatic, vertebral osteomyelitis following instrumentation of an infected urinary tract (19 cases) has revealed a total of only 5 such cases with bacteriologic proof of the same causative organism in both the urine and osseous lesions.

2. In the present case, cervical vertebral osteomyelitis with resultant quadriparesis followed transurethral resection of the prostate, and *Pseudomonas aeruginosa* was cultured repeatedly from both the urine and the osseous lesions.

3. The mechanism of the spread of infection (pyocyaneus) from the surgically

traumatized (transurethral resection) urinary tract via the prostatic venous sinuses to the vertebral venous system (by-passing the inferior vena cava with increased intra-abdominal pressure) seems to be substantiated by this case.

4. The relatively large total dose of polymyxin[B] without complication is to be noted (150 mg. per day for 22 days or a total of 3.3 gm.).

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SPONTANEOUS SUBARACHNOID HEMORRHAGE CAUSED BY EPENDYMOMA OF FILUM TERMINALE

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In 1951 Fincher[1] presented a paper entitled "Spontaneous Subarachnoid Hemorrhage in Intradural Tumors of the Lumbar Sac," based on 5 cases gathered from the literature and his personal records. The tumors in these cases, 3 ependymomas, 1 neurofibroma, and 1 neuroglioma, were manifested by abrupt onset of severe sciatica, headache, and the presence of subarachnoid blood on lumbar puncture. The similarity in the clinical picture of these cases prompted him to suggest that they represent a clinical syndrome. Roger *et al.*[4] in 1949 presented 2 cases, not included by Fincher, but closely resembling his cases. One was a "neurospongioma" (medulloblastoma) without evidence of a primary intracranial lesion, and the other a meningioma. Other authors have commented on bleeding into and around intraspinal neoplasms, giving rise to symptoms that do not fit the syndrome suggested by Fincher, and therefore not within the scope of this paper.[2,3,5,6]

Following is a report of a case of spontaneous subarachnoid hemorrhage caused by a tumor of the lumbar sac typical of the above syndrome.

CASE REPORT

A 25-year-old airman enjoyed excellent health until July 5, 1954, at which time he awoke with low backache. He had sustained no antecedent trauma other than having jumped down five steps of a stairway the day before, with no discomfort at the time. The low-back pain persisted, tending to radiate up to the dorsal region, and at times down the posterior aspect of both thighs. He was otherwise asymptomatic until July 8, 1954, when he awoke from a nap with a throbbing bifrontal headache, which persisted until admission at an Air Force Base Hospital 10 hours later. On admission there, the patient was found to be alert and oriented. Temperature was 99.2°F., pulse rate 70, and blood pressure 110/80. The only positive finding was low-back pain experienced on attempted flexion of the neck. Routine laboratory examination was normal. During the first hours in the hospital, the patient's headache became much more severe, with onset of nuchal rigidity, nausea, and vomiting. Accordingly, a lumbar puncture was done. The opening pressure was 180 mm. of water. The fluid was grossly bloody in all three specimens. The centrifuged supernatant fluid showed marked xanthochromia. Cell counts and protein determination on the spinal fluid were not done. The patient was placed on bed rest. He required narcotics for the relief of headache. The patient became afebrile on the 4th hospital day, his temperature having been in the range of 100° to 101°F. By the 5th hospital day, the patient was asymptomatic and was transferred to a neurosurgical center. Because of a mix-up in his records, he was inadvertently discharged from that center without further studies being done. Up to that time, repeated neurologic examinations had been normal.

In October 1954, his original physician discovered that he had not had diagnostic studies at the neurosurgical center, and subsequently on Oct. 21, 1954, the patient was transferred to the Lackland Air Force Hospital. He had been asymptomatic from the time of discharge until admission at that time. General physical and neurologic findings were normal on ad-

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FIG. 1. Lumbar myelogram showing the intradural tumor outlined by dye introduced both below and above it.

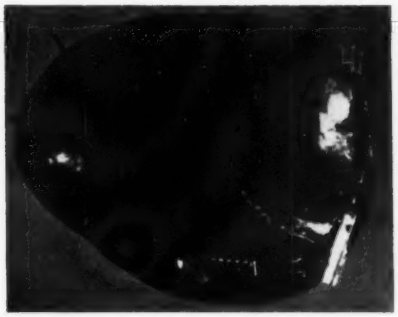

FIG. 2. The dark circumscribed ependymoma removed at surgery.

mission. On October 30, 1954, bilateral carotid angiography failed to show any abnormality. The patient was returned to duty on Nov. 1, 1954.

Shortly thereafter, he noted the return of mild low-back pain, not related to exertion. The pain increased gradually in frequency and severity, and after 4 days of outpatient physical therapy without relief he was admitted to his base hospital on Jan. 28, 1955. For several days prior to admission, the patient had noted aggravation of backache with coughing and deep inspiration. Neurologic findings were normal. Lumbar puncture showed grossly cloudy and bright yellow spinal fluid which clotted spontaneously. Jugular compression revealed no evidence of subarachnoid block. The spinal fluid contained 440 mg. per cent protein, 1840 erythrocytes, 10 lymphocytes, and 53 mg. per cent sugar. In view of these findings, the patient was transferred to the Neurosurgical Service at Lackland.

Examination. During his workup, bilateral sciatica developed, worse on the left than on the right, aggravated by cough and relieved by bed rest. He had no headache, and there were no subjective neurological symptoms referable to his bladder, lower extremities, or sexual organs. He walked with a limp because of pain in the back and legs. The neck was supple, but on flexion, low-back pain was aggravated. Jugular compression also aggravated the low-back pain. The normal lumbar lordosis was obliterated. There was tenderness to percussion over the lower lumbar spines. Flexion was limited to 45° because of pain. Straight leg raising was normal bilaterally. Neurological findings were normal except for equivocal diminution of the left patellar reflex.

Bleeding and clotting times were normal. Roentgenograms of the lumbar spine were normal. An electromyogram of the lower extremities was normal.

On Feb. 25, 1955, Pantopaque myelography was done. The needle was introduced at the lumbosacral level, and a partial block was demonstrated on jugular compression. The fluid was xanthochromic and clear and contained 500 mg. per cent protein. Five cc. of Pantopaque were introduced and an intradural block was shown at the L4 level. An additional 1 cc. of

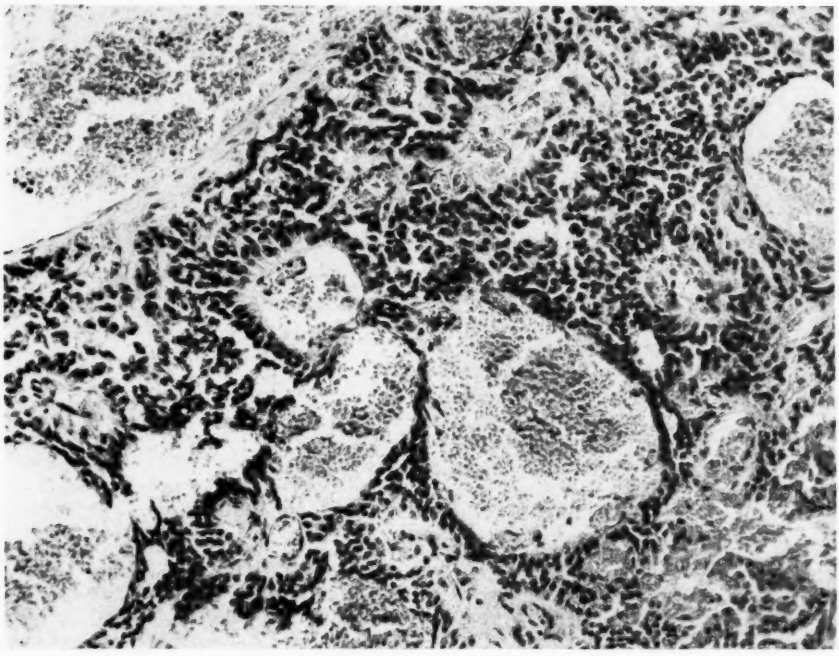

FIG. 3. Microscopic view of the papillary ependymoma showing the marked vascularity of the stroma (×180). (A.F.I.P. Acc. No. 696139)

Pantopaque was then introduced at the L2–3 level to outline the upper level of the obstruction, which was at the rostral level of L4 (Fig. 1).

Operation. On Mar. 1, 1955, lumbar laminectomy was performed. After opening the dura mater, the arachnoid was found to be markedly thickened and fiery red over the full extent of the exposure. A dark purple, ovoid tumor of the filum terminale, 4 cm. in length, was found filling the subarachnoid space (Fig. 2). After dividing the filum terminale, the tumor was lifted out with ease, since no nerve roots were attached to it.

Postoperative course was uneventful. Neurologic findings remained normal. The patient was discharged from the hospital on Mar. 16, 1955. He has continued to work and was asymptomatic when last heard of in October 1956.

Histopathologic diagnosis, confirmed by the Armed Forces Institute of Pathology, was papillary ependymoma. The marked vascularity of the stroma was noted (Fig. 3).

COMMENT

An ependymoma of the cauda equina gave rise to subarachnoid hemorrhage of such striking character as to prompt investigation by cerebral angiography. Since it is not uncommon for blood in the lumbar sac, from intracranial source, to produce low backache and sciatica, the patient's original complaint of backache was ascribed to this phenomenon. Had laboratory examination of the original specimen of spinal fluid been done and shown marked elevation of the protein, as it did later, perhaps the spinal origin of the lesion might have been suspected earlier.

This case further documents the clinical syndrome proposed by Fincher.

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MASSIVE SUBDURAL HYDROMA COMPLICATING TORKILDSEN PROCEDURE FOR POSTERIOR THIRD VENTRICLE OR PINEAL TUMOR*

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A 41-year-old white male with a high degree of choked disc bilaterally was found by ventriculography to have a tumor of the posterior part of the 3rd ventricle or the pineal body. A Torkildsen procedure was carried out and postoperative roentgen-ray therapy was given. The patient did well for 6 weeks thereafter, when headache, vomiting and marked irrationality developed. Approximately 7 weeks after the Torkildsen procedure, an enormous right-sided subdural hydroma was found and drained with a subdural catheter in the right frontal region. There was immediate relief of the disabling clinical syndrome including the subsidence of 2–3 diopters of choked disc bilaterally. He has remained well to date, 15 months after the last operation.†

This serious postoperative complication of the Torkildsen procedure, namely a massive surgically important subdural hydroma on the same side as the shunt operation, is new in our experience and is discussed with particular reference to the mechanism of its formation and its surgical relief.

CASE REPORT

E.L.W., white male, aged 41, was admitted to the Medical College of Virginia Hospital on Dec. 8, 1955. He complained of severe headache and was found to have bilateral choked disc of 4–5 D. He was of Polish extraction and his history was rather vague and inaccurate because of difficulty in language. Headache had largely subsided on Dec. 9, 1955, and the patient was anxious to return home. It was with difficulty that a ventriculogram was agreed upon. The neurological findings were normal except for the definite degree of choked discs. Examination by an ophthalmologist, Dr. E. W. Perkins, revealed no hemianopsia and normal acuity with bilateral enlarged blind spot. In July, 1955 the patient had had measles, at which time his headaches began, and it was thought temporarily that possibly he had an optic neuritis incident to the measles.

1st Operation. Ventriculography on Dec. 13, 1955, showed enormous lateral ventricles (124 cc. of fluid under pressure were removed from one lateral ventricle and a slightly less amount of air was introduced). The films revealed a mass either of the posterior part of the 3rd ventricle or of the pineal body. The posterior fossa was immediately entered through a vertical midline incision and a standard Torkildsen shunt was carried out on the right side.

Course. The patient did very well thereafter, the choked disc subsided promptly, and he was given roentgen-ray therapy. [In the following months the patient received three series of roentgen-ray treatments and by October, 1956, 10 months after the Torkildsen procedure, a total of 9,000 roentgen units (tumor dosage) had been delivered to the tumor.] The ventricular fluid Wassermann was negative, the protein being only 5 mg. per cent, with 1 cell. Flocculation test of the blood for syphilis was also negative.

The patient was discharged from the hospital on Jan. 3, 1956, his discharge having been

* Read at the 18th annual meeting, American Academy of Neurological Surgery, Phoenix, Arizona, November 10, 1956.

† This sentence was added (May, 1957) subsequent to the reading of the paper, thus allowing a considerably longer follow-up period.

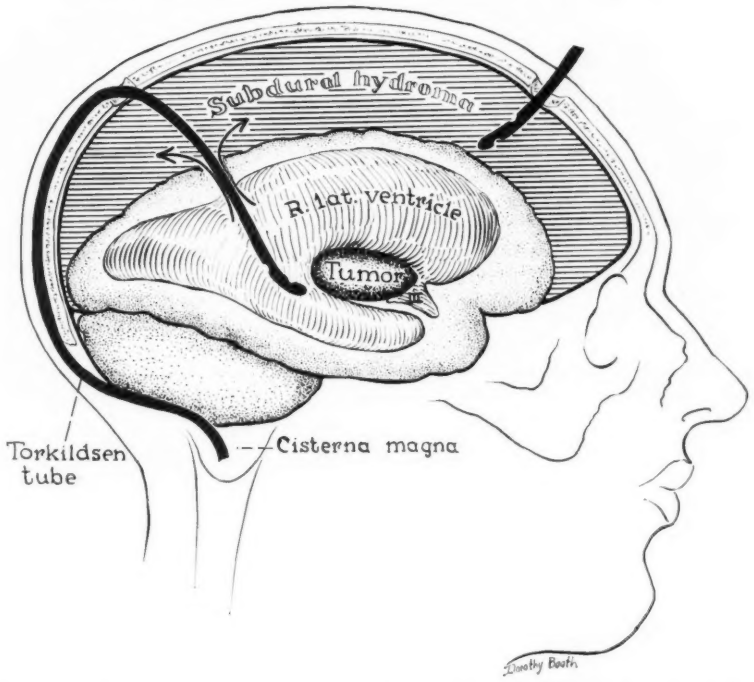

FIG. 1. Artist's conception of the two operations in this patient. The right-sided subdural hydroma is being drained by the anteriorly placed catheter left in situ for 4 days. The arrows show the presumed method of formation of the subdural hydroma. The posteriorly placed Torkildsen tube is functioning normally.

delayed somewhat by the roentgen-ray therapy. He was in good condition, with marked recession of the choked discs. He then did well for several weeks but because of a markedly irrational behavior, severe headaches, during which he held his head and moaned, and restlessness, he was admitted to Johnston-Willis Hospital, Richmond, Virginia, on Feb. 9, 1956. He had, at that time, 2–3 D. of choked disc bilaterally and because of the rather disturbing clinical syndrome, which seemed to be acute and urgent, it was determined to make frontal burr openings primarily to tap the lateral ventricles as a therapeutic measure when further roentgen-ray treatment was to be given, as it was thought that he probably had again a high degree of increased intracranial pressure with internal hydrocephalus.

2nd Operation, Feb. 11, 1956. Much to our surprise, when an anterior frontal burr opening was made on the right side, an enormous subdural hydroma was immediately encountered, the cerebral cortex and the overlying arachnoid being several centimeters from the under surface of the dura mater, so that the cortex could scarcely be visualized even when the dura mater was opened somewhat to aid removal of the fluid and the insertion of the subdural catheter (Fig. 1). The lateral ventricle on the left side also was tapped through a left frontal burr opening, the brain there being merely slack in appearance under the dura mater and the left lateral ventricle was found to be also very large. The subdural hydroma overlying the right cerebral hemisphere easily measured 80 cc. in volume.

Postoperative Course. The patient improved rapidly. A progress note on Feb. 12, 1956,

the day after surgery, stated that he was doing well; the dressings were wet with cerebrospinal fluid, especially noticeable when he coughed or cleared his throat as the uncovered protruding catheter was observed. His temperature, pulse and respiration were normal and he was alert without any headache and was also entirely rational.

On Feb. 14, 1956, the head was dressed, the dressings still being wet, and the drain was permitted to remain in position. On February 15, the subdural catheter was removed, and two interrupted black silk sutures were placed in the scalp to stop effectively the leak of fluid. He was kept on antibiotics during the time of drainage of the subdural hydroma. The patient's condition was excellent at all times after surgery. He did not remember having been admitted to the hospital on Feb. 9, 1956.

On Feb. 16, 1956, 24 hours after the subdural catheter had been removed, the head dressing remained dry. The pupils were equal, he could count fingers accurately with each eye and the visual fields grossly were full and wide. He still had, however, about 2 D. of choked discs at this time. He was alert and rational, moved all of his extremities equally well, was ambulatory, and his temperature was normal.

On February 21, the day of discharge (all the scalp sutures having been removed), the incisions were well healed per primam. The optic disc margins were beginning to come into view with perhaps ½ D. elevation at most. The temperature was normal and he was ambulatory about the ward.

Laboratory Studies. On admission the patient had 4.85 million red blood cells per c.mm., 12.5 gm. of hemoglobin (85 per cent), and 7,800 white blood cells per c.mm., with 78 per cent polymorphonuclear cells. The ventricular fluid from the left anterior horn on February 11 showed only 1 lymphocyte and 12 mg. per cent of protein; Kolmer reaction was negative. Unfortunately, the clear, colorless subdural fluid (hydroma) evacuated at this operation was not examined in the laboratory. His blood pressure varied from 150/90 to 102/64 during his hospital stay. Roentgenograms of the skull on Feb. 17, 1956, showed only the Torkildsen tube in normal position on the right side and evidence of the suboccipital decompression on the right side that had been done in December, 1955, at the time of the initial surgery (Torkildsen procedure).

Subsequent Course. On March 3, 1956, the patient returned to the office. His general condition was excellent. The operative wounds were well healed, there was no headache, he was ambulatory, the optic discs were slightly hazy without measurable choking, and he had *no subjective complaints of any kind*, approximately 3 weeks after drainage of the hydroma.

He has been seen on several occasions since that time, the last examination being on May 17, 1957 (15 months after drainage of the subdural hydroma and 17 months after the original Torkildsen procedure) when he was doing his regular work in the mill where he was employed. The optic discs showed no choking, he had no headache, diplopia, ataxia, or syncope, or subjective complaint of any kind, and apparently the Torkildsen tube is working well. From the objective standpoint, no abnormality could be demonstrated neurologically except for moderate nystagmus on far lateral gaze, bilaterally.*

COMMENT

This case represents a postoperative complication that we had not seen in our clinic before; namely, a massive subdural hydroma of surgical importance on the same side as the Torkildsen tube which came on in less than 2 months after the Torkildsen procedure was performed for a pineal tumor or a mass in the posterior part of the 3rd ventricle. In trying to determine the pathologic physiology accounting for this lesion one can think only that the Torkildsen tube acted not only as planned, i.e. to short-circuit ventricular fluid around the posterior part of the 3rd ventricle and the pineal region (the site of the tumor) into the cisterna magna,

* This paragraph was added (May, 1957) subsequent to the reading of the paper in order to supply a considerably longer follow-up report.

but also the tube *may have functioned as a wick* allowing ventricular fluid in a "relaxed" brain on the right side to well up around the catheter into the subdural (extra-arachnoid) space in large amounts. Thus the cerebral hemisphere on the right side became markedly compressed by the time of drainage of the subdural hydroma, approximately 2 months after the Torkildsen procedure. One wonders also why the subdural hydroma did not refill again shortly thereafter as the same conditions with the Torkildsen catheter in situ still prevail at the present time. However, it has now been 15 months since the drainage of the subdural hydroma and the patient is in normal condition, working regularly at his occupation in a paper mill with no evidence of the disturbing clinical syndrome that he had when the hydroma was drained.* Probably the brain (right cerebral hemisphere, especially) has re-expanded to some extent and the opening in the cerebral hemisphere has so sealed off that now the Torkildsen tube is carrying out only its *desired* function of draining fluid from the lateral ventricular system directly to the cisterna magna. Presumably, also, the *midbrain tumor was extremely radiosensitive*, allowing the ventricular block to be relieved at an early date; this would also facilitate, perhaps, the formation of the subdural hydroma.

SUMMARY

A case is presented of a patient with a presumed neoplasm of the posterior part of the 3rd ventricle or pineal body (verified by ventriculography) who initially responded well to a Torkildsen procedure and roentgen-ray treatment. Later a large, subdural hydroma developed over the right cerebral hemisphere with very disturbing symptoms relieved completely to date by simple catheter drainage of the hydroma. The apparent mechanism of formation of this complication is briefly discussed.

* This sentence was inserted (May, 1957) subsequent to the reading of the paper in order to supply a considerably longer follow-up report.

NOTICES

TEACHENOR MEMORIAL

A memorial fund has been established to honor the late Doctor Frank Randall Teachenor who not only helped to pioneer the specialty of neurological surgery but was widely known as a teacher, physician and a great humanitarian. Under the organization of a Teachenor Memorial Committee contributions are being received to build a Frank Randall Teachenor Memorial Operating Theater in the new Research Hospital Medical Center at Kansas City, Missouri. It was decided the fund, while national in scope, should be applied to a facility that would maintain a working memorial in the community that Doctor Teachenor served so well and unselfishly.

The Committee hopes, through this publication, to reach friends who might wish to participate in establishing this memorial. Anyone wishing to do so may make a contribution to the Frank Randall Teachenor Memorial Fund Committee in care of the Research Hospital, 23rd and Holmes Streets, Kansas City, Missouri.

REPORT OF *Ad Hoc* COMMITTEE ON GRANT SUPPORT OF TRAINING IN NEUROLOGICAL SURGERY

At the invitation of Dr. Pearce Bailey, Director of the National Institute of Neurological Diseases and Blindness, a meeting of leading authorities in the field of neurological surgery was held in Bethesda on February 24, 1958.

In attendance were: Dr. Howard Brown, Assoc. Clinical Professor, Neurosurgery, University of California; Dr. Winchell Craig, Professor Emeritus of Neurological Surgery, Mayo Foundation Graduate School, University of Minnesota; Dr. Leonard Furlow, Assoc. Professor, Clinical Neurosurgery, Washington University Medical School; Dr. Franc D. Ingraham, Assoc. Professor of Surgery, Harvard University; Dr. Bronson S. Ray, Professor of Clinical Surgery (Neurosurgery), Cornell Medical College; Dr. Henry G. Schwartz, Professor of Neurosurgery, Washington University Medical School; Dr. Harry Wilkins, Professor of Neurosurgery, University of Oklahoma. Also in attendance were: Doctors Bailey, Masland, Seger, Merritt, Sahs (Chairman of the NINDB Neurology Training Grant Committee), and Farber (Executive Secretary, Neurology Training Grant Committee).

This group was convened at the suggestion of the National Advisory Neurological Diseases and Blindness Council, to discuss and to make recommendations concerning the role of the Institute in augmenting neurosurgical scientific manpower as an important measure to facilitate carrying out the Institute's responsibility for the development and maintenance of a broad program of research in the field of neurological and sensory disorders.

Following a clear presentation by Dr. Bailey of the provisions of Public Law 692 of the 81st Congress, and a review of the development and objectives of the NINDB, Dr. Craig presided over the session. After lengthy discussion which took into consideration the various forms of training support available, the group was unanimous in reaching the following conclusions:

1) Although there is no clearly recognized shortage of practitioners of neurological surgery, there is a continuing need for qualified teachers and investigators trained in the discipline of the specialty, who will devote themselves to academic careers.

2) The goal of promoting the development of future teachers and investigators

in neurological surgery can be achieved within the framework of existing programs established by the NINDB, reference to which is made in the following paragraphs.

3) NINDB Special Traineeships, a program established to support advanced training for those who have completed the residency requirements or their equivalent in a specialty and wish further preparation for careers as teachers and investigators, have been and continue to be available to promising neurological surgery candidates. The encouragement of young men in training to enter the academic field is primarily the responsibility of their mentors. Such intellectual encouragement to enter upon an academic and research career in neurological surgery can be fortified economically by the use of Special Traineeships since such awards provide adequate stipends.

4) To encourage young men to pursue investigation prior to completion of residency requirements, the utilization of the existing program of NINDB Postdoctoral Research Fellowships should be borne in mind. The stipends provided under this program will support an individual while carrying out a specific research project or while spending a period of time in the laboratory over and above the usual period assigned during the residency program.

5) After considerable discussion, the *ad hoc* committee concluded that the award of program grants to institutions to support training during the residency period, does not represent a general need in neurological surgery at the present time.

The foregoing conclusions of this committee shall not be construed as being in any sense retroactive so far as previous NINDB commitments for support of training in neurological surgery are concerned. Furthermore, it is recommended that these conclusions be reviewed and re-evaluated after a period of two years.

GERMAN SOCIETY OF NEUROSURGERY AND SWISS SOCIETY OF NEUROSURGEONS

A combined meeting of the German Society of Neurosurgery and the Swiss Society of Neurosurgeons will take place in Zürich, Switzerland, July 25–26, 1958, in the Kantonsspital, Rämistr. 100. Members of American and European neurosurgical societies are cordially invited to attend. Information is obtainable from Prof. Dr. H. Krayenbühl, Neurochirurgische Universitätsklinik, Kantonsspital, Zürich. Reservations for accommodations in Zürich should be made through Mr. René Mayer, Reisebüro Kuoni, Zürich, Bahnhofsplatz.

The subjects of the program are: (1) Otfrid Foerster Memorial Lecture and presentation of the Otfrid Foerster Medal to Prof. Dr. L. van Bogaert, Berchem-Antwerp; (2) Glioblastomas of the cerebrum, and (3) miscellaneous papers.

HARVEY CUSHING SOCIETY

The 26th annual meeting of the Harvey Cushing Society was held at the Mayflower Hotel, Washington, D.C., April 21–23, 1958. The following officers were elected: President, Bronson S. Ray, New York; President-Elect, James L. Poppen, Boston; Vice-President, James W. Watts, Washington; Secretary, David L. Reeves, Santa Barbara; Treasurer, Hendrik J. Svien, Rochester, Minn.; Historian, Louise Eisenhardt, New Haven; member of Executive Committee, Howard A. Brown, San Francisco.

Members appointed from the Society to the American Board of Neurological Surgery are Donald D. Matson, Boston, and Edwin B. Boldrey, San Francisco.

Mrs. Dean H. Echols, New Orleans, is President of the Ladies Auxiliary, and Mrs. Edmund J. Morrissey, San Francisco, is President-Designate.

The next meeting of the Society will be in New Orleans, Louisiana, at the Roosevelt Hotel, April 30–May 2, 1959.

LATIN AMERICAN CONGRESS OF NEUROSURGERY

The 8th Latin American Congress of Neurosurgery and the 4th Congress of Electroencephalography and Clinical Neurophysiology are scheduled to be held from April 12–15, 1959 in Santiago, Chile. The main topics will be cranial nerves and cerebellum. Specialists from Argentina, Bolivia, Brazil, Colombia, Mexico, Peru and Uruguay have been assigned as responsible authors of these topics.

There will also be an International Symposium under the chairmanship of Dr. A. Earl Walker, Baltimore, Maryland, U.S.A.: 1) Ultrasonics; 2) Depth Recording; 3) Cerebral Circulation; 4) Ultrastructure of Cortical Neurones.

The Congress of Electroencephalography will deal with the following subjects: 1) Electromyography; 2) EEG abnormalities in cases of tumors of the floor of the third ventricle.

WESTERN NEUROSURGICAL SOCIETY

At the 3rd annual meeting of the Western Neurosurgical Society in Reno, Nevada, September 29–October 1, 1957, the following officers were elected: President, Howard Brown, San Francisco; Vice-President, Rupert Raney, Los Angeles; Secretary-Treasurer, Ernest Mack, Reno. The 4th annual meeting of the Society will be held at the Del Monte Lodge, Pebble Beach, California, October 20–22, 1958. Attendance is for members and invited guests.

MEETINGS

American Academy of Neurological Surgery, Royal York Hotel, Toronto, Canada, Nov. 6–8, 1958. Dr. Eben Alexander, Jr., Bowman Gray School of Medicine, Winston-Salem, N.C., Secretary.

American Association of Electromyography and Electrodiagnosis, Jefferson Medical College, Philadelphia, Pa., Aug. 24, 1958. Dr. George H. Koepke, University Hospital, Ann Arbor, Mich., Secretary.

Association for Research in Nervous and Mental Disease, Hotel Roosevelt, New York City, Dec. 12–13, 1958. Dr. Rollo J. Masselink, 700 West 168th St., New York 32, N.Y., Secretary. (Subject: Neuromuscular disorders.)

Congress of Neurological Surgeons, St. Francis Hotel, San Francisco, Calif., Oct. 30–Nov. 1, 1958. Dr. Richard DeSaussure, Jr., 525 Physicians & Surgeons Bldg., Memphis 3, Tenn., Secretary.

German Society of Neurosurgery, Kantonsspital, Zürich, Switzerland, July 25–26, 1958.

Harvey Cushing Society, Roosevelt Hotel, New Orleans, La., April 30–May 2, 1959. Dr. David L. Reeves, 316 West Junipero St., Santa Barbara, Calif., Secretary.

International Congress of Neurological Surgery (II), Washington, D.C., Oct. 14–20, 1961. Dr. Bronson S. Ray, 525 East 68th St., New York 21, N.Y., Secretary-General.

Latin American Congress of Neurosurgery, Santiago, Chile, April 12–15, 1959. Dr. Alfonso Asenjo, Casilla 70 D, Santiago, Chile, Secretary General.

Neurosurgical Society of America, The Homestead, Hot Springs, Va., March 4–7, 1959. Dr. Frank P. Smith, 260 Crittenden Blvd., Rochester 20, N.Y., Secretary.

Scandinavian Neurosurgical Society, Copenhagen, Denmark, August 29–30, 1958. Dr. Bendt Broager, Neurokirurgisk afd., Bispebjerg Hospital, Copenhagen NV, Denmark, Secretary.

Southern Neurosurgical Society, Americana, Bal Harbor, Miami Beach, Fla., Jan. 23–24, 1959. Dr. C. D. Hawkes, 22 N. Manassas St., Memphis 5, Tenn., Secretary.

Swiss Society of Neurosurgeons, Kantonsspital, Zürich, Switzerland, July 25–26, 1958. Dr. H. Krayenbühl, Neurochirurgische Universitätsklinik, Kantonsspital, Zürich, Switzerland.

Western Neurosurgical Society, Del Monte Lodge, Pebble Beach, Calif., Oct. 20–22, 1958. Dr. Ernest W. Mack, 505 So. Arlington Ave., Reno, Nev., Secretary.

JOURNAL OF NEUROSURGERY

The *Journal of Neurosurgery* is devoted to the publication of work relating primarily to neurosurgery, including studies in clinical neurophysiology, organic neurology, ophthalmology, radiology and pathology. Short articles on unusual cases and on technique, special instruments or equipment that might be useful to others in the field are also acceptable.

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